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SOME PRACTICAL ASPECTS OF ENDOCRINOLOGY.[1]

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THE subject of endocrinology is a very important one, but over it there is a great deal of controversy. It is, of course, impossible to deal with more than a very small fraction of the subject in a lecture, so I have chosen to attempt to deal with certain practical aspects in which I am particularly interested.

The function of the endocrine glands is to provide certain specific chemical substances, essential for the normal life of those cells of the body which in the process of evolution have sacrificed the ability to manufacture such substances for themselves. These chemical bodies are poured direct into the blood stream, hence arises the name ductless glands. If the body is to be normal and in health, the quantity of these substances or hormones, as they are called, must be of certain definite amounts. It is a proven fact that the hormones from one gland greatly influence the action of the hormones from other glands. In other words the products of the endocrine glands form a balanced system which may to some extent be considered analagous to the strands of a cobweb. Each strand of the web will represent a product of an endocrine gland and the general character of the web will be due to the resultant of the various strains exerted by the individual threads of the web. With any one thread some of the other threads are synergic, others are antagonistic.

Now if all the threads are acting healthily, we have a normal individual, but the possibilities of variation of stresses and strains should one or more threads be altered, are almost unlimited, even in so simple a structure as a cobweb. Hence we can appreciate the complexity of possibilities in endocrinology. For example, if the strain of any individual thread is increased or decreased, every other thread has its tension altered in one or other direction and the result will vary as to whether the change in tension of the first thread to go wrong was during the making of the cobweb (that, is congenital abnormalities) or after the web was finished (acquired abnormalities). To a very large degree each of us is individualistic according to the balance of his endocrine glands and we each tend towards a certain endocrine type. Starting with a normal individual, all grades of variation in a given direction could be picked out from the general population up to a person who would have to be considered as pathological; for example, we could start with a perfect specimen of an adult male six feet high and then pick out all intermediate stages up to a person suffering from gigantism. This tendency of an individual towards an endocrine type is partly congenital and partly due to the effects various infections have had on the endocrine system. Infection undoubtedly plays a determining part in the glandular make-up of many persons and we all know people whose type has been almost fundamentally changed by the effects of typhoid fever, diphtheria or other infection. The milestones in life also leave their mark on our glandular balance and infections during puberty and the menopause often lead to profound results.

What is the chemical nature of the various glandular products? This has only been ascertained for two glands, epinephrin, the product of the adrenals and thyroxin from the thyreoid gland. Both of these products have now been prepared by synthesis.

Now let us briefly review the chief clinical types of glandular anomalies. The products of three glands, the pituitary, the gonads and the thyreoid gland, play the chief part in determining the stature and general body structure as a whole.

The Pituitary Gland.

The pituitary gland exerts the greatest influence on growth and for the most modern knowledge on this aspect of endocrinology I would refer you to Harvey Cushing's two articles in *The British Medical Journal*, July 2 and 9, 1927. He deals very concisely with conditions of hypopituitarism, such as Simmond's disease and various forms of nanism and then with acromegaly.

I would like to say something of the condition of dyspituitarism or Fröhlich's disease. By Fröhlich's disease is understood a disorder of metabolism affecting children or adolescents in which pronounced obesity, overgrowth of long bones and a variable degree of hypoplasia of the genitalia are associated. The ætiological factor may be either a congenital abnormality of the pituitary gland, neoplasm of the pituitary gland or the pituitary region of the brain or *sequelæ* to infections.

In the last named the infection is most often accompanied by a fever with hyperpyrexia and the sequelæ are probably due to hæmorrhage or infarction. Figure I illustrates a typical patient with Fröhlich's disease. This patient was perfectly normal till the age of seven, when he became affected by pneumonia, then measles, then bronchopneumonia in quick succession. That the pituitary gland is primarily at fault has been proved experimentally in animals, by operative removal of tumours in man and clinically the great improvement that may follow the administration of suitable preparations of pituitary extract, is strong evidence in favour of a pituitary lesion being the cause of the condition.

The next two illustrations (Figures II and III) are those of a very remarkable patient in whom the lesion of the pituitary gland was associated with depressed thyreoid function, basal metabolic rate of –29% and severe diabetes. This patient failed to respond to treatment, probably owing to the presence of large multiple abscesses which occurred in many parts of the body.

The Gonads.

The gonads play important parts in the body economy, besides insuring the next generation. Loss

[1] A British Medical Association Lecture, delivered before the Central Southern Medical Association, New South Wales, on September 9, 1927.

FIGURE I.
Patient with Fröhlich's Disease.

of their internal secretion results in a condition of eunuchoidism. When the loss is at all severe and occurs early in the life cycle of an individual, a very typical picture is produced. We have a person all "skin and bone." The height may or may not be excessive, but the normal proportion of the length of the trunk to the legs and the span to the height is lost. Normally the measurement symphysis to soles and symphysis to vertex should be exactly the same and the span exactly equal to the height. A eunuchoidal type of person has a symphysis to soles measurement five to ten centimetres (two to four inches) greater than symphysis to vertex and the span is five to ten centimetres (two to four inches) greater than the height. Such individuals show many other stigmata of their condition. The complexion is sallow and "muddy," the skin is wrinkled, parchment-like, but soft, the face is babyish, but old at the same time. The teeth are

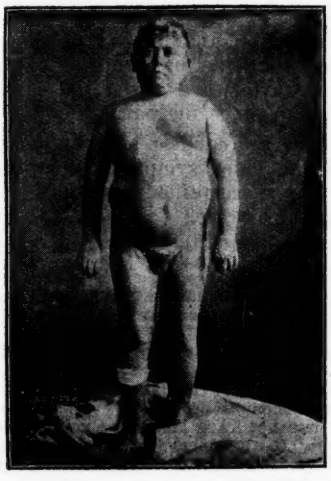

FIGURE II.
Patient with Lesion of Pituitary Gland, Depressed Thyreoid Function and Diabetes, Front View.

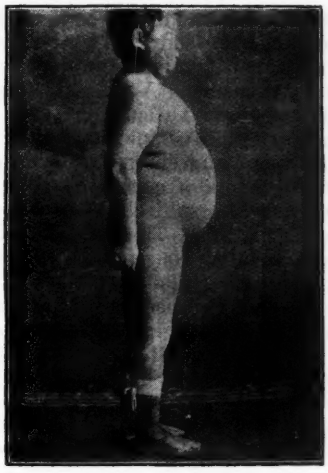

FIGURE III.
Patient with Lesion of Pituitary Gland, Depressed Thyreoid Function and Diabetes, Lateral View.

large and generally well preserved. In females there is no breast development, in the male no secondary hirsuties. In the male the voice is high pitched. Such patients do not have good health, are generally easily tired and senescence is premature. Figure IV illustrates the physical stigmata of a typical patient who was thirty years old.

I do not intend to deal with the pineal gland, the adrenals, the thymus or parathyreoid at all, not that these glands are unimportant, but because time does not permit.

The Thyreoid Gland.

Now let us turn our attention to the thyreoid gland. This may be compared to a queen controlling all the squares and pieces of the body. No pawn moves or stays, but is activated by her influence

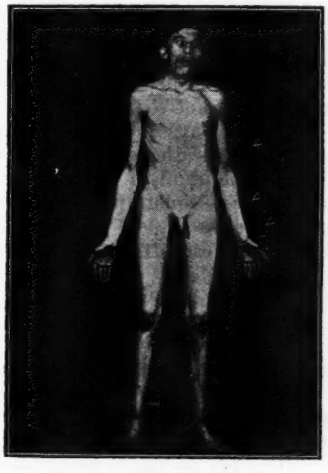

FIGURE IV.
Patient with Gonadal Deficiency.

and when with indolent ease she neglects her throne, lethargy pervades the body, all activities languish, the State becomes poor and miserable; we have the condition of myxœdema. A patient with advanced hypothyreoidism or myxœdema presents a remarkably constant symptom-complex, the chief symptoms being loss of "pep" and energy, everything becomes a burden, there is loss of memory, irritability, slowness in movement, subjective feeling of cold, obstinate constipation. The physical signs are a characteristic bloated coarseness of the face, the skin of which becomes alabaster-like and somewhat translucent, in some respects wax-like; the hair of the head is dead and wig-like, generally coarse and often has become dark, the eyebrows are scanty, often absent in the lateral half and what hair there is, is short and has a singed appearance. The expression is dull and apathetic, movements are slow and deliberate, there is a considerably increased latent period in performing a voluntary act after the word of command. The skin of the body feels cold and almost like a corpse, it is dry and rough, resembling a leather-jacket's skin. The patient may or may not be stout, some severely hypothyreoid patients may be quite wasted as the extreme anorexia which is sometimes present results in the intake of very little food. The voice is pathognomonic, slow, thick, monotonous, throaty, unpleasant and produced as if the tongue were in the way. The pulse is often not slow, as the heart is not efficient; the circulation rate is slow, but as the heart does not contract vigorously it has to contract fairly frequently, some seventy to eighty beats a minute, whereas in normal people under basal conditions the rate is often under sixty. The severity of the condition is best determined by estimating the basal metabolic rate. This will be discussed a little later on.

The treatment, of course, is to give thyreoid extract orally or, as is being done in some places, thyroxin intramuscularly or even intravenously. In using the dry thyreoid extract a good plan is to start with 0·12 gramme (two grains) daily and increase by 0·06 gramme (one grain) every week until the patient appears quite normal. The dose should then be reduced. About 0·24 gramme (four grains) daily of an active preparation should be sufficient to keep a patient within normal limits. The best way to see if the dose is adequate, is to repeat the basal metabolism estimation when the patient has been on a constant dose for from three to four weeks. A dose of 0·06 gramme (one grain) of thyreoid more or less often makes a tremendous difference to the patient's well-being. Unfortunately there is no standard of potency for thyreoid extract in Australia. Certain brands apparently have very little thyroxin present. It is unfortunate that we have so far neglected to standardize this therapeutic agent. If excess of the extract is being given, the patient begins to suffer from palpitation on exertion, by being "nervy" and by somewhat choreiform twitchings of the extremities; the pulse rate will be found to be rapid, from ninety per minute upwards.

No Roman emperor ever kept his slaves in more abject terror than does the thyreoid when the queenly sceptre is wielded with a tyrannous hand. A spirit of apprehension prevails everywhere. Every cell is working feverishly and the whole body gives the appearance of constant fear. I shall not go into every symptom and sign of exophthalmic goître, but will outline my routine method of history taking at the Royal Prince Alfred Hospital, where we attempt to get comparative data from every case in order to assess the relativity of the degree of excess of thyroxin *per se* and degree of stimulation of the nervous system. I have given up any attempt to divide cases into such groups as exophthalmic goître, toxic-adenoma, parenchymatous goître, hyperthyreoidism *et cetera* because we have not been able to confirm clinical diagnoses with the histological findings. Dr. Tebbutt has pointed out very clearly how often a swelling which clinically would be diagnosed as an adenoma, is nothing more histologically than a hyperplastic portion of the gland, without any capsulation and quite indistinguishable from other portions of the gland; while on the other hand a gland which clinically is uniformly enlarged and of uniform consistency, often contains a deep-seated encapsulated adenoma which could not be diagnosed by palpation. We have not been able to confirm Plummer's ideas on adenomata of the thyreoid and the only classification which I think is justifiable, in the present state of our knowledge is as follows:

1. Hyperthyreoidism in which with the signs and symptoms attributable to excess of thyroxin production there is what may be regarded as a proportional stimulation of the nervous system.

2. Hyperthyreoidism with nervous manifestations out of proportion to the degree of thyreoid stimulation.

3. Hyperthyreoidism in which the nervous manifestations are relatively less than would be expected from the degree of thyreoid overaction.

The examination and history of the patient are so arranged that the information obtained therefrom can be afterwards used in classifying into which group to place the patient.

In all cases the earliest sign or symptom to be noted and the chief symptoms complained of by the patient are ascertained and the history of any possible exciting cause.

Next, answers to the following questions are obtained:

Thyreoid: How long has swelling been present? Has it varied in size? Is there any sensation of choking or fullness in the neck?

Heart: Is there palpitation, shortness of breath or swelling of feet?

Cerebral Symptoms: Has the patient changed in disposition? Is he emotional or "nervy"? Are purposeless movements present? Are lashings-about noticed?

Vasculatory Disturbances: Are there any subjective feelings of heat or cold? Is flushing present? Is perspiration noticed?

Gain or Loss of Weight: Has either been noted?

Appetite: What is its state?

State of the Bowels: Do they act regularly?

Susceptibility to Fatigue: Is the patient easily fatigued?

Periods: Have any changes taken place since the onset of the disease?

Eyes: Are the eyes prominent? How long have they been so?

Hair: Has the character of the hair altered?

Voice: Has the character of the voice changed?

Sore Throat: Is there any history of sore throat?

Onset of Disease: Is there any relation between onset of disease and childbirth, menopause, period of stress, infection *et cetera.*

In the physical examination the patient is examined as far as possible in the anatomical position throughout.

The thyreoid is examined for the following points:

Size, shape, consistency, whether uniform or nodular, vascularity to palpation and to auscultation, presence or absence of bruits.

Eyes: Staring or not? Degree of exophthalmos? Is von Graefe sign present or not? Is any widening of the palpable fissure present?

Eyebrows: If scanty or not.

Hands: Hands are examined for tremor, the patient is made to hold both hands stretched out with the fingers separated. If the tremor is at all definite, it can be felt; false tremors generally cease when the hand is held lightly by the examiner. Note whether the hands are warm and moist. Note presence of trophic changes in the hands as regards absence of fat in the fingers. Note changes in the nails. Hyperextensibility is often marked in young exophthalmic patients.

Heart: Note the presence of tachycardia, forcible slapping first sound, murmurs, fibrillation.

Throat and Teeth: Examine the throat and teeth if indicated.

A note is always made if the patient is fidgety or lashing herself about. The hurried speech is very characteristic, the words tumble over each other on their way out.

Now let us balance these findings into the groups as to whether they are primarily due to excessive thyroxin or to overstimulation of the nervous system:

Group I: Signs and symptoms which may be attributable to both causes—palpitation, fatigue, flushings, trophic changes.

Group II: Nervous manifestations—eye signs, tremors, changes included under cerebral symptoms, changes in periods and in the voice.

Group III: Due to excessive thyroxin production. Under this heading come the physical findings of the thyreoid and the feeling of warmth to the examining hand, the loss of weight in spite of a good appetite, subjective feeling of heat, perspiration, increase in metabolism as measured by the metabolic rate.

Of all the evidence of increased thyroxin production, a raised metabolic rate is of outstanding importance and it is to be regretted that a method of calculating the degree of nervous stimulation on a mathematical basis is not yet available.

Now let us briefly consider the principle of basal metabolism. Every living cell is constantly undergoing metabolic changes which for the most part are thermal in nature. The total production of heat from the metabolism of all the cells of all the tissues cannot be reduced beyond a certain minimum which is attained by reducing the subject to basal conditions. This is achieved clinically by keeping the subject at complete rest and without food. The heat production of a subject under such basal conditions can be calculated by measuring the gaseous exchange. The total heat production is then expressed in terms of calories per square metre of body surface per hour. When we express the difference in the number of calories produced as a percentage variation from the number of calories that should be produced by a normal person of the same age and sex, we obtain what is known as the basal metabolic rate. With a careful technique this can be obtained with considerable accuracy.

Why are estimations of the metabolism of so much value in thyreoid disorders? It is because the rate of metabolic activity of every cell in the body is practically controlled by the amount of thyroxin in circulation. No other catalyst of cellular activity is found in the body under normal conditions. Of course, rises in temperature lead to increased cellular activity, just as they lead to increases of any other chemical reaction; therefore one of the first considerations before the basal metabolic rate is estimated is that the temperature must be normal. In hyperthyreoidism the basal metabolism may be raised as much as 100% in extreme cases. In most severe cases there is an elevation of the metabolism up to 80%. In hypothyreoidism the basal metabolic rate is lowered to from –20% to –50%.

When a diagnosis of hyperthyreoidism is made, how is the patient to be treated? The first consideration is complete rest. This of itself may bring the metabolism down a considerable extent. The rest should be made as congenial as possible and mental rest must be assured. During this period of rest Lugol's iodine should be given from 0·6 to 2·4 mils (ten to forty minims) *per diem.* This will have its maximum effect in from ten to fourteen days and seldom does any more good after that. Another estimation of the metabolism should now be carried out and if a considerable drop has taken place, hemithyreoidectomy should be performed without further delay. If the metabolism has not fallen, further rest and sedatives may be tried, until a drop in the metabolism indicates that the patient is suitable for operation. Patients who are operated on with a rising metabolism, seldom do well. It has been our experience that if the degree of hyperthyreoidism has been at all severe, removal of half the gland is the minimum that may effect a cure, and that often a second operation will be required, even when hemithyreoidectomy has been performed. This will be rendered evident by a failure of the metabolism to remain within normal limits for any appreciable time after the first operation.

In lieu of an operation X ray therapy may be used. Results from this form of treatment have in our experience been very inconstant. A good result seems to depend on adequate dosage which should be controlled by an expert. Some of our patients have given extraordinarily good results. In other cases it has failed. All surgeons to whom I have

spoken, agree that X ray therapy makes subsequent operation more difficult.

Prolonged medical treatment in my opinion is very disappointing and very seldom effects a cure. So long as the patient is resting, the metabolism may remain low, but a return to the patient's home environment almost invariably leads to a relapse within a shorter or longer period. I have come to this conclusion against my will, for it seems the greatest pity that the only way to restore a patient to a useful life is by the sacrifice of a large portion of so important an organ.

In the treatment of hyperthyreoidism the question of food intake is of great importance. An adequate mixed diet is essential. The increase in metabolism means increase in fuel consumption and loss of weight means that the body is burning its own tissues. The best way to insure an intake of food of high caloric value is to give fat in an easily digested form, for example, butter and cream. It is a great mistake to cut down protein foods, as the nitrogen loss is increased in hyperthyreoidism.

Septic infections undoubtedly play a large part in the production of hyperthyreoidism, so that every effort should be made to eradicate septic foci.

There are just a few other points worthy of discussion.

The effect of hyperthyreoidism on menstruation seems to vary with locality. In London at Saint Thomas's Hospital practically every patient with hyperthyreoidism had either amenorrhœa or irregular, scanty periods. At the Royal Prince Alfred Hospital patients very seldom gave a history of a changed menstrual cycle, unless the disease was very severe.

In regard to the bowels, in England constipation was almost the rule. In Sydney the bowel action is generally quite regular. Diarrhœa is uncommon in both localities.

The chief symptoms in the majority of cases are: Complaint of being "nervy" and "shaky," palpitation and shortness of breath, loss of weight, loss of energy, headaches.

Less frequently the patient seeks attention for choking feelings, eye signs or a swelling in the neck.

Diabetes.

Now let us turn our attention to another disease of endocrine origin, namely, diabetes.

Experimentally, diabetes is produced by total or sub-total pancreatectomy and the disease results from the loss of the products of secretion from the β cells of the islets of Langerhans. The condition of hyperglycæmia and acidosis resulting therefrom can be remedied by the exhibition of "Insulin." In human diabetes the injection of "Insulin" subcutaneously also remedies the hyperglycæmia and allows the oxidation of the fatty acids to end-products to take place, but nevertheless the pathology of human diabetes is not yet satisfactorily explained in all cases. *Post mortem* the pancreas of a person dead of diabetic coma may appear normal both to macroscopical and microscopical examination. The discovery of "Insulin" has not perfected our knowledge of the pathology of the disease, though it has given us a most potent therapeutic agent. However, as the object of this address is mostly clinical, we cannot deal further with the pathology of the disease, but must turn our attention to the modern means of its investigation.

In most cases the suspicion of diabetes is aroused by the presence of a reducing substance in the urine. With the nature of possible reducing substances in the urine we are not concerned, because glucose is always to be suspected when on boiling an equal quantity of urine and Fehling's solution there is a prompt and definite reduction of the copper salt to the oxide. However, if the reduction is doubtful and the urine is concentrated, the urine should be diluted one in one with water and if reduction still takes place with this diluted urine, there is every reason to assume that the reducing substance is glucose.

When the urine has been proved to contain sugar, can a diagnosis of diabetes be definitely made? In many cases the reduction is so definite and the presence of acetone so pronounced and the history of great loss of weight, of polyphagia, polydypsia and asthenia so definite and the sunken cheeks, dry parched lips with their layer of inspissated mucus so characteristic that there is no doubt of us having to deal with a case of severe diabetes.

On the other hand, there is a number of cases which is larger or smaller in proportion to the number of patients whose urines are examined as a routine, in which the suspicion of a mild diabetes is aroused by the presence of a reducing substance in the urine.

How are we to decide definitely the significance of the presence of sugar in these cases? Ten years ago there was no way available to decide this important problem, but the advent of microchemical methods of blood sugar estimation has given us a clinical test by means of which we can determine the degree of impairment to the glycogenic function or definitely prove an innocent glycosuria. Our ability of being able to appraise definitely the impairment or otherwise of the glycogenic function depends on determining the changes in the percentage of glucose in the circulation which takes place after the ingestion of a large quantity of sugar. In the normal fasting individual there is present in the capillary blood from 0·08 to 0·12 gramme of glucose *per centum*. After the ingestion of fifty grammes of glucose in one hundred cubic centimetres of water the blood sugar promptly rises, so that at the end of half an hour it is normally 0·165% to 0·185%. Then there is a gradual fall and by an hour and a half the original level is reached. A small secondary rise often then ensues. This rise and fall in the blood sugar does not depend on the rate of absorption of sugar from the alimentary tract, as there is still a large amount of sugar present in the stomach at the end of two hours and absorption is still proceeding rapidly. The mechanism of the rise and fall of the blood sugar

is not understood, but its occurrence depends on the presence of an intact glycogenic function. The diabetic has lost to a greater or less extent the ability to bring into play the mechanism we have just discussed, so that there is not the same control over the level of the blood sugar. A diabetic patient who has not been receiving treatment has a fasting blood sugar from 0·2% to 0·4% and after the ingestion of glucose the level of sugar in the blood continues to rise and the degree of severity of the diabetes may be gauged by the type of curve that ensues.

In cases of innocent glycosuria the blood sugar curve after the ingestion of glucose is always within normal limits, indeed it often tends to be lower than normal and yet sugar will appear in the urine. We are accustomed to think of a level of the blood sugar above which sugar will appear in the urine. This is called the renal threshold and for practical purposes may be taken as 0·18%. If sugar appears in the urine with the blood sugar below this threshold of 0·18%, we have to deal with a case of renal glycosuria. The amount of sugar in the urine in these cases is never very large, seldom more than 1%. The fact that after a large dose of glucose there is very little sugar in the urine does not necessarily mean that we are dealing with a case of renal glycosuria, because old-standing diabetics who have an advanced arteriosclerosis, develope a condition in which the renal threshold is far above the normal one of 0·18%. Indeed, cases of diabetes occasionally come under our notice in which after the ingestion of glucose the blood sugar rises to as much as 0·4% and yet no sugar appears in the urine.

In considering the severity of a case of diabetes we must balance with the sugar intolerance the degree of ketosis. These are by no means proportionally severe in all cases. As a general rule the more acute the disease and the younger the patient, the greater the degree of ketosis, while the more chronic and older cases are characterized by a milder acidosis in proportion to the degree of sugar intolerance. It is always of great importance to have some idea of the extent of the acidosis present. Clinically this is best ascertained by examining the urine for the presence of acetone and the Rothera test is by far the most satisfactory method for testing.

Having decided on the degree of severity, how is the patient to be treated? Will "Insulin" be required or not? Certain cases are so severe that it can be readily seen that "Insulin" will be required if the patient is to receive an adequate intake of food for maintenance purposes. However, it is best more or less always to begin on a set plan except in cases of coma or threatening coma. Another exception is mild cases which are discovered indirectly. The plan to adopt except in these exceptional cases is as follows.

Principles of Treatment of Diabetes.

The following principles of treatment of diabetes have been modified from Campbell.

1. Determine the caloric requirements for a basal diet:
   (*a*) Calculate surface area from height and weight of patient using a Du Bois chart.
   (*b*) Calculate from the basal chart caloric requirements per square metre of body surface (Aub-Du Bois).

CALORIC REQUIREMENT PER SQUARE METRE OF BODY SURFACE (AUB-DU BOIS.)

Age (in Years).	Males. (Calories per Day.)	Females. (Calories per Day.)
10–12	1,236	1,200
12–14	1,200	1,116
14–16	1,104	1,032
16–18	1,032	960
18–20	994	912
20–30	948	888
30–40	948	876
40–50	924	864
50–60	900	840
60–70	876	816

Then from (*a*) and (*b*) estimate the total basal caloric requirements.

2. The amount of protein to be given is the first thing to fix and this is determined on the basis of one gramme of protein for each kilogram of body weight for all patients except children, when 1·5 grammes per kilogram should be given or in emaciated subjects when twenty grammes more than calculated should be given.

3. From the following formulæ then determine first the required composition of the diet:

(*a*) $C = 0{\cdot}024M - 0{\cdot}41P$

(*b*) $F = 4C + 1{\cdot}4F$

where C = carbohydrate in grammes.
P = protein in grammes at rate of one gramme per kilogram of body weight.
F = fat in grammes.
M = daily basal metabolic requirements in calories.

Then determine the glucose value and the fatty acid value of the diet from equation:

(*a*) $G = C + 0{\cdot}58P + 0{\cdot}1F$.

(*b*) $FA = 0{\cdot}9F + 0{\cdot}42P$.

This should give a ratio of glucose to fatty acid of 1 to 2.

We determine the glucose, fatty acid ratio or the keto-antiketogenic ratio, as it is called, because as Woodyat showed, the combustion of fat requires the simultaneous combustion of glucose in the ratio given above to avoid acidosis. If, as happens in some cases, acetone still appears in the urine on such a diet, a rebalance of the diet is necessary. If the urine is sugar free, increase the glucose. If glucose is present in the urine, increase the "Insulin."

With the patient at rest feed the above basal diet for several days. Estimate quantitatively the sugar excreted in the twenty-four hour sample of urine. If the patient becomes sugar free on this diet, increase it and give carbohydrates five grammes and fat twenty grammes. It is desirable to determine the blood sugar in order to ascertain whether increases may be safely given or not. Further similar increases in diet may be made after the pancreas is allowed to rest for five to fourteen days. After three such increases in diet it is advisable to increase the protein eight grammes and fat ten grammes. Gradually increase the exercise as the caloric allowance increases. Excessively high caloric diets are never indicated.

If patient does not become aglycosuric on a basal diet "Insulin" should be used. The grammes of glucose excreted determine the initial day's dose of "Insulin" in units. Usually it is 1·5. Give the "Insulin" in two portions daily, thirty minutes before breakfast and supper. As the patient's urine becomes sugar free, reduce the dose by one-third or increase diet as above. Establish the patient on a definite diet with known "Insulin" dosage and normal blood sugar levels.

Hypoglycæmia results from unsuitable administration of "Insulin" in point of time or amount, vomiting and diarrhœa, failure of food absorption, over exercise and increase in the natural tolerance for carbohydrate. Immediate carbohydrate administration is the cure. The carbohydrate, as oranges, syrup or candy, is usually administered by

mouth in the less severe cases. In the most severe cases of hypoglycæmia, when the patient is unconscious, an intravenous injection of glucose is necessary.

Diets for diabetics should be built up from a basal level to a maintenance level. For practical purposes, the maintenance level may be calculated as a 50% addition to the basal level.

For people on heavy occupations a more liberal addition must be made.

The following is an example:

Mr. X., *ætatis* forty-five years, weighed sixty-five kilograms (ten stone). His height was 162·5 centimetres (five feet five inches). Then from the Du Bois chart the surface area was 1·75 square metres. From the caloric requirement chart a male *ætatis* forty-five requires 924 calories per square of body surface *per diem*. Therefore this patient requires 924 × 1·75 calories = 1,617 calories.

Weight (ten stone) = 65 kilograms. Therefore protein required = 65 grammes = 260 calories.

Then carbohydrate = 0·024 × 1,617 − 0·41 protein = 38·8 grammes.

And fat = 38·8 × 4 + 65 × 1·4 = 155·2 + 91 = 246·2 grammes.

Therefore this is the diet on which to start the patient and we should work up to a maintenance diet of $^3/_2$ times this, that is, 2,426 calories.

In coma or threatened coma a concentrated solution of glucose from 10% to 30% should be given intravenously along with large doses of "Insulin," from twenty to fifty units, straight away to be repeated in two to three hours according to indications. Blood sugar estimations are of the greatest assistance in controlling the dose of "Insulin." In mild cases a quantitative diet of from 2,200 to 2,500 calories in which a limit is placed on the amount of carbohydrate intake, is all that is required. After the diet has been standardized, it is generally sufficient to keep the urine sugar and acetone free, but it is preferable to make periodic estimations of the fasting blood sugar level. Patients who are receiving "Insulin," when estimations of the blood sugar are not available, should for safety's sake have a little sugar in the urine.

It has not yet been shown that any form of treatment can effect a cure or lead to a definite increase in the tolerance for sugar. Apart from diet, attention should be given to the eradication of septic foci. Great care should be taken to tell the patient to go to bed with any slight infection, such as a sore throat or a boil, as such conditions seem greatly to upset the glycogenic function. It is often a good plan to increase the "Insulin" during any feverish condition or if the patient is not on "Insulin," to cut down the diet.

In conclusion I would like to stress the point that it is the acidosis which kills, not hyperglycæmia, so that in treating a patient with diabetes the urine must be kept free from both sugar and acetone.

SOME COMMON DISORDERS OF THE FEET.[1]

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THE list of disorders to which the human foot is liable, is a lengthy one and consideration of many of them is by no means free from complexity. It is not possible in a short paper to do more than touch very lightly on some of the essential facts of symptomatology, pathology and the treatment of the commoner forms of foot disorders. I must ask your indulgence, therefore, if I pass lightly over some aspects and omit mention of some varieties of foot troubles which you may have expected me to dwell on at some length.

I have thought it possible that any discussion which may follow this paper may serve to enlighten me on these deficiencies and perhaps allow me to repair them in some measure. Some of the views and methods of treatment I will put before you are strictly personal and therefore controversial, but I do not propose to make any statement or claim which personal experience has not substantiated.

Complaints put forward by patients who suffer from foot troubles may be fairly readily classified under one or a combination of the following heads: (i) Pain, (ii) deformity, (iii) impairment of or loss of function.

Pain is the symptom which most frequently leads the patient to seek relief, but as in all types of orthopædic work it is important to find out the actual symptom the patient seeks to have relieved. Examination of a foot may reveal a gross deformity, whilst the patient's complaint is of the pain associated with an insignificant-looking callosity which is but a secondary effect of the deformity. If the latter be corrected short of rendering the further growth of the callosity impossible, treatment will have failed.

Corns.

Corns or results of continuous chafing pressure are classified commonly into hard and soft varieties and I believe the two types are held by some to arise from separate causes. There is an idea that because soft corns are frequently found in juxtaposition on two contiguous toe surfaces, they are of infective origin. Personally I have not seen a corn, whether hard or soft, which did not have as its sole cause the two elements of friction and pressure.

There is no need to argue the case for the hard corn. The familiar reaction to a new pair of boots, the corn-capped angulations of contracted toes are too familiar and their cause too obvious to need discussion and yet in the case of the soft corn a similar cause is just as obvious, if sought for.

The commonest site for soft corns is at the very apex of the cleft between the fifth and fourth toes, a site which at first glance would appear to be one of the most secluded and free from pressure in the body. But an examining finger will find that this is not the case and beneath each corn will be found the resistance of hard bone. In effect such corns arise as the result of pressure of the head of the first phalanx of the fifth toe against the head of the fourth metatarsal or base of the first phalanx of the fourth toe. Sometimes the soft corns are distal to this situation, but in every case I have

[1] A British Medical Association lecture delivered at a meeting of the Central Southern Medical Association, February, 1928.

found a bony projection beneath each apposing corn. I have seen a bursa directly beneath one of the corns in the cleft between these two toes.

A very common sequence is to find a curled up contracted little toe with a large painful corn on its most prominent angle and soft corns in the cleft. In these cases a direct line of pressure can be made out and if the foot be held firmly and pressure inwards be exerted on the exterior corn, the whole train of painful symptoms is elicited. Sometimes the pain radiates along the course of the short saphenous nerve.

Corns and callosities occur on the sole frequently as the result of faulty weight bearing, associated either with impaired function, as in flat foot, or with deformity, as in contracted toes or claw foot.

Care must be taken to distinguish corns from warts. The latter may occur on the sole of the forefoot, either singly or in definite outcrops. When they are single they are sometimes very difficult to differentiate, particularly since such a wart may be capped with a definite callosity. If the latter be carefully pared away the distinctive appearance suggestive of a wart is seen and very little further paring will result in profuse bleeding.

Treatment of Corns and Warts.

The cause is generally found to be either (i) faulty footwear, (ii) errors of foot function in walking and standing and (iii) deformity. Correction of deformity by manipulation or operation and in the case of soft corns, when conservative measures fail, I remove one of the offending bony projections, preferably the head of the fourth metatarsal. This gives permanent relief.

If operation or manipulation be contraindicated or refused, a metatarsal bar or a crooked and elongated heel will often alleviate the symptoms of corns or callosities on the sole.

For removal of the actual corn salycilic acid pigment and pumice stone, judiciously applied, are most effective.

For warts an application of radium or X rays may be used.

Ingrowing Toenails.

Ingrowing toenails result in most cases from cramped and faulty footwear. If prophylactic means are insufficient, I think it best to remove the whole nail. Apply a small rubber band as a tourniquet to the base of the toe and carefully dissect out whole of root of the nail.

Hammer Toe.

The deformity may be fixed or the toe may be capable of passive extension. The condition is seen in its most typical form in the second toe. It is usually without subjective symptoms until a corn forms either on the dorsum of the first interphalangeal joint or the distal end of the toe or both situations. Sometimes the only complaint is of pain beneath the head of the second metatarsal, when it will be found that the hyperextended first phalanx has become subluxated at its base on to the head of the metatarsal, thus forcing the latter plantarwards and causing pressure, a callosity and pain.

Treatment.

Under no circumstances amputate. A wedge of skin and bone including the corn should be exsected at the interphalangeal joint, the deformity at the metatarso-phalangeal joint corrected and the toe straightened and kept on a splint until bony union occurs between the two phalanges.

Contracted Toes.

Pain often accompanies the deformity of contracted toes and is caused by the resultant corns and callosities. One of the most usual to contract is the little toe. It is sometimes found displaced dorsally at the metatarso-phalangeal joint and folded upon itself at the interphalangeal joint. A painful corn is usually present on the dorsum of the angle formed by the latter contracture. Pain is often described running up the side of the foot and pressure over the corn will often elicit pain along the course of the short saphenous nerve.

Treatment.

I am averse to amputation. If the displacement at the metatarso-phalangeal joint is undone, then the corn does not reform and the pain ceases. Remove a diamond-shaped area of skin from the dorsum of the foot proximal to the toe and divide all contorted soft tissues, including the collateral ligaments if necessary, and flex the toe at the metatarso-phalangeal joint; fix into position in splint and later with strapping. I use a simple aluminium splint cut to fit the foot and find it very effective.

Dorsal Contraction of the Toes.

Teece and myself have described independently a condition, by no means rare, in which the toes, frequently including the great toe, are dorsally contracted. No element of claw foot or other deformity is present in this condition.

The condition in my experience is much more common in males and of the patients seeking relief all have been in the early twenties.

In the fully developed condition the patient is quite crippled. There is acute pain on walking, related to large callosities beneath the forefeet and the toes are folded up on the foot (the base of each first phalanx is subluxated on to the corresponding metatarsal head) and do not make contact with the ground at any stage of walking or standing.

Patients in the earlier stages describe discomfort and pain on walking. I am not able to account for the ætiology, though certain physical signs constantly observed would lead me to believe that the condition represents a stage in development of a local muscular dystrophy.

Treatment.

In the early stage a metatarsal bar and a moulded splint to the sole serves at least to check deformity.

In later stages radical division of all contracted structures with six weeks in splints in over-correction is necessary.

Claw Foot.

Contracted toes are also a feature of the condition known as claw foot. Here the greatest degree of the deformity is seen in the great toe. There is a dropping or pulling down of the forefoot through contraction of the plantar fascia and the gradual increase of the deformity throws more and more weight on the forefoot.

The treatment of this condition is aimed more at the deformity of the foot than of the toes and a flattening out of the foot by division of the superficial contracted structures or by stripping the intrinsic muscles off their origin from the *os calcis* with transplantation of the *extensor longus hallucis* into the first metatarsal head will give relief when the condition has not fully developed.

Bunions.

It will save much time if we consider the question of bunions in connexion with the deformities with which they are most frequently associated and the first of these is *hallux valgus*. This condition occurs undoubtedly as a congenital deformity and is not infrequently seen in young children. It has been my experience that in nearly every case the children are females.

It may be argued with some reason that the cases seen in adults (and here again women predominate) are really congenital in origin, but the fact remains that the wearing of short and tightly fitting shoes with pointed toes will accelerate, if it does not actually produce, the condition.

In young children there can be no question but that the primary deformity occurs at the first tarso-metatarsal joint. There is a *varus* deformity of the first metatarsal which allows the toe to be readily displaced outwards at the metatarso-phalangeal joint. Bursæ are rarely seen; there may be some thickening about the metatarso-phalangeal joint and the chief complaint is of slight pain and the deformity.

Treatment.

Correct the displacement of the metatarsal by removing a wedge base outwards from near the proximal end of the shaft; divide the collateral ligament on the outer aspect of the metatarso-phalangeal joint. Correct the deformity, splint and put the patient into shoes and stockings as described below.

In adults the condition is seen most typically: the grossly displaced toe, projecting exostosis, capped with a bursa inflamed sometimes to the point of suppuration.

In old people whose activities are mild, it is sufficient to chisel off the exostosis, round off the cut bone edges and dissect out the bursa in order to give them relief. Care must be taken to see that the urine is free from sugar and that the circulation is adequate in this type of case.

In vigorous and active adults correction of deformity and restoration of function must be aimed at.

Arthroplasty after removal of the metatarsal head and division of the tendons of the *extensor longus hallucis* only when the latter is definitely contracted give very excellent results. Do not remove sesamoids.

Any operation which does not have as its result correction of deformity and a useful range of active movements at the new joint, cannot be considered as successful. Commence active and assisted active movements on the eighth day after operation and gradually increase them; the splint should be worn in the intervals and every night for six months; digitated stockings and slippers fitted with a felt bar should be worn and then square-toed shoes are essential. A small tag of leather beneath the great toe joint prevents the weight from falling on the second metatarsal head.

Bunions are also found fairly frequently on the dorsum of the first metatarso-phalangeal joint in the condition known as *hallux rigidus* which results from the onset of osteoarthritis of the joint. Usually the osteophytic outgrowths typical of this condition are exhibited about the margins of the articulating bones, causing an interlocking and a limitation of movement often with the joint in slight flexion. In some cases one or more osteophytes become more prominent on the dorsum of the metatarsal head and the friction of the shoe on the outstanding skin covered exostosis causes a bunion.

If movement is not greatly restricted it may be sufficient to chisel off the exostosis; otherwise removal of the metatarsal head is called for. The operation is more difficult to carry out than in the case of *hallux valgus*, as there is not the same amount of tissue available for a flap as in the latter condition.

Painful Heels.

Pain frequently occurs at the back of the heel. It may be due to inflammation of the bursa beneath the *tendo Achillis* or in young subjects to an epiphysitis of the post-epiphysis of the *os calcis* comparable to Osgood-Schlatter's disease of the tibial tubercle. In gonorrhœal subjects there is often found an enlargement of the bone at this situation and in some cases the pain is caused finally by the patient continuing to wear the same shoes or boots which fitted him before the enlargement of the bone occurred.

Make the patient rest, raise the heel of the boot twelve millimetres (half an inch), remove the bursa and give pin-firing with red hot needles. These measures are most likely to give relief.

Most commonly, however, pain is complained of beneath the heel and examination will reveal tenderness of varying intensity over one or both tubercles of the *os calcis*.

Pain in this situation is found in persons suffering from gonorrhœa, from flat foot, in people of overweight and as a result of trauma.

Skiagrams will reveal in most cases the presence of a spur of bone running forward from the tubercle in the direction of the fibres of those intrinsic muscles of the foot which take origin from this process.

In spite of the textbook advice to remove these spurs as a means of alleviating the pain, it is very doubtful if the spurs or exostoses actually are in all cases the primary cause of the latter. Rather I think that the cause of the pain is that set of pathological circumstances which gives rise to the spurs themselves.

There is plenty of clinical evidence to support this view. Radiographs show that many cases of spur of the *os calcis* exist without any complaint from the patient and at operation no active projection of the spurs towards the sole can be made out; they are hidden away in the substance of the muscles.

If operation is undertaken, it has to be remembered that what looks like a spur in the radiogram, is actually a shelf of bone projecting forward from the tubercles. Wide exposure by turning the tissues of the heel forward is the best method of attack and allows total removal.

Bursæ are found at times deep in the muscles and may be of considerable extent and must be totally extirpated.

Prolonged rest with or without operation offers the best promise of cure in this difficult condition. Bursæ should be removed.

In cases associated with flat foot a Whitman's brace hollowed out to relieve pressure on the painful area may be of great value.

Flat Foot.

In a short paper it is impossible to traverse anything more than the bare outline of the subject of flat foot and I must content myself with a few general principles.

The term flat foot is a very unfortunate one. It is unscientific and focuses attention upon one of the minor aspects of a condition in which the actual shape of the foot may be of but scant importance.

The high arch and narrow waist of the so-called civilized foot is merely a reflection of the wasting due to loss of function of the great muscles which lie along the inner border and under surface of the sole. Even in its attenuated state the *abductor hallucis* is a solid piece of musculature, as are also the *flexor brevis digitorum, adductor minimi digiti* and *flexor brevis hallucis.* In non-booted peoples they are fully developed and in consequence the shape of the foot in those peoples is flat according to our standards and yet who can avow that flat foot in such peoples is pathological?

My own view is that the determining factors by which we decide whether or not so-called flat foot exists, is not the shape of the foot, but the line along which the body weight is transmitted through the tibiæ to the feet.

If the body weight is transmitted straight down the tibiæ with the thigh in a neutral position between internal and external rotation, it will rest on the foot squarely and the latter will take the weight and transmit it to the ground through its natural points of contact, namely, flat of heel, outer border of foot and forefoot and toes.

If the thigh is rotated inwards or outwards it will tilt the foot inwards or outwards into a *valgus* or *varus* position as the case may be.

This, then, may be said and proved by demonstration to be the test of correct foot reaction in relation to weight bearing and to constitute the only sure test as the presence or otherwise of "flat foot."

For my own convenience I have classified this condition into physiological and anatomical types. In the former it will be found that all parts of the foot work normally, there is no fixed deformity and the body weight can be voluntarily aligned on the feet through the correct axis. In the anatomical form the foot is fixed in deformity from various causes: (i) Paralysis of the *tibialis posticus* and/or *tibialis anticus.* (ii) Spasm of the peronei. (iii) Inflammatory changes in the tarso-tarsal or tarso-metatarsal joints. (iv) Trauma, Pott's fracture, fracture of the tarsal bones. (v) Congenital abnormalities of the scaphoid and astragalus, congenital absence of the fibula. (vi) Changes in the shape of the bones of the leg as in bow leg, where the accompanying internal rotation of the lower end of the tibia swings the body weight over on to the inner border of the foot, producing a condition of so-called flat foot which is very difficult to deal with. (vii) *Talipes equinus,* acquired and congenital.

Symptoms.

The symptoms include deformity fixed or capable of voluntary correction with or without pain, aching, tenderness about the tubercle of the scaphoid and over the inferior calcaneo-scaphoid ligament. Pain is absent during rest. The condition may be acute or chronic.

Treatment.

The sole aim of treatment is to have the body weight transmitted down the tibiæ square into the foot. In the physiological type this is effected as follows: If the condition is acute, give rest with foot inverted and adducted in plaster until all pain and tenderness have disappeared, then fix the foot in inversion by means of adhesive strapping, crook the heels and give exercises to develope all foot and leg muscles and those of the thigh, especially the external rotators. The patient is shown how to walk correctly and must learn to do it voluntarily and unconsciously. Do not give supports in this type, especially in children.

In the anatomical type restore correct alignment by wrenching, operation, plaster *et cetera* cast for Whitman's brace with the foot in the corrected position, exercise and movements. Walking is valuable and should be carried out daily as a routine. Heel and toe exercises should not be given at the onset, particularly in patients whose flat foot is the result of muscle weakness.

REMARKS ON FLAT FOOT.

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Postural flat foot is a general term for a number of clinical states due to breakdown of the normal mechanism for maintaining the shape and posture of the foot. These states are known as: (i) foot strain, (ii) *pes valgus*, (iii) *pes plano-valgus*, (iv) *pes planus*, (v) flattening of the transverse arch and metatarsalgia. The foot is sometimes flat congenitally or as a result of paralysis or of osseous or arthritic lesions; these forms are comparatively uncommon and are not within the scope of this article.

Anatomy and Physiology.

The functions of the foot are: (i) to support the weight of the body in standing, (ii) to raise and propel the body by leverage in walking. For the first function the foot is constructed on the architectural principle of intersecting longitudinal and transverse arches. To subserve the second function, a joint is provided at the ankle and multiple joints throughout the foot which by giving resiliency and flexibility to the structure, increase its efficiency as a lever and absorb shocks.

The longitudinal arch has the strong rigid mass of the *os calcis* for its posterior pillar; but anteriorly, where flexibility is required, it is segmented, consisting of an outer part composed of the cuboid and outer two metatarsals and an inner, more mobile part, comprising the scaphoid, cuneiform and inner three metatarsals. The highest point of the inner part is the astragalo-calcaneo-scaphoid joint; of the outer part, the calcaneo-cuboid articulation.

The transverse arch is most evident at the tarso-metatarsal joints. Forward of this it gradually diminishes in height, until at the level of the metatarso-phalangeal joints it forms a low arch known as the anterior transverse metatarsal arch.

The astragalus can scarcely be said to enter into the formation of the arches: it rests upon the upper surface of the *os calcis* and takes part in the astragalo-calcaneo-scaphoid articulation; thereby it distributes to the arches the body weight it receives through the tibia.

The natural shape of the foot is determined by the formation of the bones of the arches. The posture of the foot, on the other hand, is determined solely by the activity of its muscles. The shape of the normal foot varies considerably in different individuals between the limits of long and narrow and short and broad. The height of the longitudinal arch is by no means constant: certain individuals have naturally a high arch, others a low arch.

It is often erroneously stated or inferred that the arches of the foot are supported by ligaments. In the foot as elsewhere the purpose of ligaments[1] is merely to hold the articulating bones in apposition and to check momentarily excessive movement. Being inelastic, ligaments cannot withstand continuous strain, under which they soon stretch. The ligaments of the foot are strongest on the plantar aspect—the concavity of the arches—so as to check sudden depression of the arches. Although many and complicated, they can be resolved[2] into short tie-bands between the segments of the arches and long bands connecting the pillars. Thus, for the longitudinal arch the short bands are the long and short plantar and the inferior calcaneo-scaphoid ligaments and the plantar ligaments of the several joints; the long tie is the plantar aponeurosis. For the transverse arch the short ties are the transverse fibres of the plantar ligaments of the tarsal and metatarsal joints and the transverse metatarsal ligament; long ties are supplied by the tendons of *peroneus longus* and *tibialis posticus*.

The maintenance of the arches, when weight is borne upon the foot, depends normally upon the long and short muscles of the foot. During standing the body weight tends to dorsiflex and pronate the foot. This effect, if allowed, would strain and depress the inner part of the longitudinal arch and "lock" the joints by moving them to their full extent in one direction. For rapid change from the standing position, however, it is essential that the joints should be maintained in a "neutral" position. Accordingly, those muscles which can resist dorsiflexion and balance the foot in the mid-position between supination and pronation, are found during standing to be tonically contracted, that is, to be exhibiting postural tone. These muscles are in general those of the back of the leg. In preventing dorsiflexion, the triceps is chiefly concerned, aided by the long flexors of the toes, the peronei and *tibialis posticus*. The mid-position between supination and pronation is maintained by the balanced activity of the peronei on the outer side and *tibialis posticus* and the long flexors of the toes on the inner side. *Tibialis posticus* also forms a sling under the summit of the inner part of the longitudinal arch while *peroneus longus* acts similarly for the outer part. The latter also indirectly supports the inner part by holding the great toe firmly against the ground. The long flexors of the toes and the muscles of the sole—in particular the lumbricals, interossei and *abductor hallucis*—are also tonically contracted, helping to maintain the arches and keeping the toes pressed against the ground.

In normal standing the superimposed weight is borne mainly by the heel, the outer border of the sole and the heads of the metatarsals, especially the first and fifth. When the weight is placed upon the foot, small adjustments of the position of the joints take place by which the position most favourable for weight-bearing is attained. The outward result of these adjustments is that the foot broadens slightly and the internal border which in the resting

foot is slightly concave inwards, becomes more straight.

In normal walking, the foot acts as a flexible lever raised by the plantar-flexing muscles upon a fulcrum beneath the heads of the metatarsals. When the foot leaves the ground the tone of the plantar-flexing muscles is inhibited, while tone is manifested in the anterior muscles, which maintain the foot in dorsiflexion as it passes forwards. In correct walking the muscles play an important part in preserving the normal conformation of the foot; but when a faulty method of walking is adopted, leverages are changed, the action of certain muscles is impaired and alteration of conformation follows.

The movements of the foot are dismissed somewhat cursorily in anatomical and surgical textbooks; but to understand the deformities of the foot which may arise from deficiency of the postural mechanism, it is essential to know clearly what the movements are and where they take place.

Of the foot as a whole there are four movements, namely, (i) plantar flexion (extension), (ii) dorsiflexion, (iii) pronation (abduction-eversion), (iv) supination (adduction-inversion). Plantar flexion and dorsiflexion are confined almost entirely to the ankle joint; the foot can be dorsiflexed until it forms with the leg an angle less than a right angle by 10° to 20°; plantar flexion can proceed until the dorsum of the foot is in line with the front of the leg. At the mid-tarsal and tarso-metatarsal joints a few degrees of movement in these directions are also possible. In some persons the foot, though otherwise normal, cannot be dorsiflexed beyond the right angle; these persons are unable in walking to rise completely on to the toes and tend to push off from the inner side of the foot.

Supination and pronation are combined movements, respectively of adduction and inversion and of abduction and eversion. They are the results of changes of position simultaneously at the sub-astragaloid joint and the mid-tarsal joints (calcaneo-cuboid and astragalo-calcaneo-scaphoid joints). The shape of the articular surfaces of these joints is such that the movement of inversion (rotation inwards of the foot upon its long axis) cannot take place without adduction(displacement of the foot to the inner side of the long axis of the leg) or eversion without abduction. The chief site of the movements is at the subastragaloid joint about an axis which runs obliquely from the inner side of the neck of the astragalus to the infero-external part of the *os calcis*. Thus the movements take place below the level of the astragalus. Supination is greatest when combined with full plantar flexion, pronation with full dorsiflexion, the range between the two extremes being about 50°.

In full plantar flexion a few degrees of pure abduction and adduction are possible, occurring at the ankle joint because of the lateral play permitted when the narrow posterior part of the astragalus lies in the relatively wide interval between the malleoli.

At the metatarso-phalangeal joints flexion of the toes is accompanied by adduction and dorsiflexion by abduction. The former movement increases, the latter diminishes the height of the anterior metatarsal arch.

At the metatarso-phalangeal joint of the great toe there is normally a range of movement from 5° of plantar flexion to 45° of dorsiflexion. When this range is diminished, the individual is unable in walking to rise on to the great toe and is compelled, as with limited range of dorsiflexion at the ankle, to push off from the inner side of the foot; the bracing action of *flexor hallucis longus* upon the longitudinal arch is thus impaired and the foot is subjected to an abnormal pronating leverage. The range of abduction and adduction at the first metatarso-phalangeal joint is small in adults of boot-wearing races; but the normal range which is seen in young children and in adults who have not worn boots, is considerable and, in walking, abduction (deviation of the great toe from the mid-line of the foot) accompanies elevation of the foot on to the toes.

Ætiology.

Postural flat foot may arise in three ways:

1. When postural activity is deficient throughout the body, the condition being associated with other postural deformities, such as round shoulders and knock-knee. (The cause of generalized postural deficiency is uncertain, but mental fatigue and ill-health from focal and other infections appear to be important factors).

2. From long-continued local strain exhausting the postural reflex.

3. From inhibition locally of the postural reflex by some painful condition, such as sprain or fracture ("traumatic" flat foot) or gonorrhœal or rheumatoid arthritis ("inflammatory" flat foot). In the production of traumatic and inflammatory flat foot there are probably two factors, namely, inhibition of the postural reflex by pain and damage to bones and joint structures altering the normal conformation and alignment of the joint. According to Bankart[3] the former is the more important.

Conditions of local strain are the commonest causes of postural flat foot. These are (i) rapid increase of body weight, adding to the burden borne by the feet; (ii) prolonged standing, especially in a faulty attitude and when unbroken by periods of free activity; (iii) incorrect use of the feet in walking; (iv) footwear which gives insufficient support, or by ill-fitting interferes with the movements of the foot; (v) conditions, such as limitation of dorsiflexion at the ankle or of the great toe, which diminish the normal range of movement and impose a faulty mode of walking; (vi) corns and other minor lesions of the foot which induce protective but faulty modes of standing and walking; (vii) mal-union of fracture of the leg, ankle or tarsus, deflecting the body weight to the inner side of the foot.

Clinical Features.

The variety of forms of flat foot, indicating different reactions of the foot to the force of gravity

when the postural mechanism is deficient, is explainable probably by (i) the age of the patient at the time of onset, (ii) the type of foot, (iii) local conditions determining the site of maximum incidence of strain.

It is to be noted that pronation of the foot with flattening of the longitudinal arch is physiological in the infant. The postural reflexes of the skeletal muscles are imperfectly developed at birth, but appear during the first few years of life. The postural reflex of the muscles of the foot is being established as walking is begun. Pronation is soon corrected and the longitudinal arch raised, the foot thereby acquiring the normal conformation and posture. In some children in whom the postural mechanism is slow to develope or is delayed in development by ill-health, pronation and flattening of the foot persist, constituting the so-called "infantile" form of flat foot. This is distinguishable from congenital flat foot in which flattening is almost invariably accompanied by calcaneus or calcaneo-valgus deformity, by being supple and by a simple passive movement reducible instead of irreducible.

The form of flat foot known as *pes valgus* ("weak" or *valgus* ankle) is especially common in late childhood and early adolescence and in the slender type of individual. It is frequently associated with generalized postural deficiency. In this form chiefly the muscles supporting the subastragaloid joint appear to be deficient. The unsupported ligaments stretch and allow displacement of the *os calcis* beneath the astragalus. The foot is thus pronated at the subastragaloid joint without lowering of the arches. The internal malleolus is prominent and the foot turned outward, apparently below the malleoli, but really below the astragalus. *Pes valgus* frequently remains for long reducible by a passive or voluntary movement.

Among adolescents and adults the common forms of flat foot are *pes plano-valgus* and *pes planus* with or without flattening of the anterior transverse metatarsal arch; they are more often due to a local than to a general cause.

Foot-strain, classified as one variety of flat foot, is in a sense misnamed, since in it the arches are not depressed. It is the earliest stage of breakdown of the postural mechanism and the precursor of the conditions in which flattening of the arches has actually occurred. The patient complains of aching under the arches and of tenderness on pressure over the plantar ligaments, particularly the inferior calcaneo-scaphoid. These symptoms indicate that the muscles are inefficiently supporting the arches and that in consequence the ligaments are being strained. The symptoms are often most acute, as might be expected, in persons with a naturally high longitudinal arch. The conformation, posture and movements of the foot are normal. Foot strain is sometimes an acute disorder ("acute" flat foot), the pain and tenderness being severe and preventing walking or examination of the foot and accompanied by vaso-motor phenomena—vaso-dilatation, œdema and sweating. This condition is liable to appear in persons who return too soon after an illness to an occupation necessitating much standing, and in those who suddenly change their occupation from a sedentary to an active outdoor one.

Pes plano-valgus ("pronated" foot) is the result of neglected foot strain, but it may develope so slowly that the symptoms of strain are not manifest. It is noteworthy that rapid stretching of the ligaments is the cause of the pain in flat foot. For this reason no pain is felt with the "infantile" form, in which the longitudinal arch is undeveloped, nor in slowly-forming *pes valgus, plano-valgus* or *planus;* nor is pain felt in *pes plano-valgus* or *planus* once the deformity is fully developed. In these states, it is the deformity itself or clumsiness of gait which causes the patient to seek advice.

Pes plano-valgus is a state of persistent pronation of the foot. As stated above, the natural tendency of gravity in standing is to pronate the foot. When the postural tone of the muscles is insufficient to counteract this, the position of pronation is adopted and persists. Furthermore, the position is soon exaggerated by stretching of the ligaments on the convex aspect, allowing inward displacement of the astragalus upon the *os calcis;* it may become fixed ("rigid" flat foot) by adaptive changes, at first in the soft tissues, later in the bones.

Depression of the longitudinal arch is the least important element of the deformity. Primarily merely apparent and due to pronation, it is later real when the ligaments have stretched, permitting the bones to be displaced. But even then, the pronation element which impairs the mobility and leverage function of the foot, remains the more important.

It is usual to describe three stages in the development of *pes plano-valgus*: a first in which the deformity is correctable by a voluntary effort, a second in which reduction is partially prevented by adaptive tissue changes, and a third in which changes in the bones as well as the soft tissues fix the deformity. It may be noted, however, that the sequence is not invariable; the first stage may persist indefinitely.

Patients with *pes plano-valgus* of rapid onset may exhibit the phenomenon of fixation of the deformity by spasm of the peronei. Peroneal spasm is probably a reflex, the stimulus arising in the strained ligaments of the inner side of the foot. It can be temporarily overcome by a steady passive supinating movement, but returns when the surgeon's grip is released.

Not uncommonly in *pes plano-valgus* pain is felt in unexpected situations. Thus pain may be felt over the tip of the external malleolus when it impinges upon the everted *os calcis.* Aching up the outer aspect of the leg and in the hip and back is sometimes complained of, pointing to the general disturbance of function caused by the faulty posture of the foot. Vasomotor disturbance is frequent and significant of the functional origin of the deformity.

In a few patients flattening of the longitudinal arch is a more evident feature than pronation. This is the condition of *pes planus*. Probably persons who acquire *pes planus*, have a naturally low arch or one that because of postural deficiency is depressed or does not develope in early childhood. Thus the bones and joints are adapted to the abnormal position early in life and with break-down of the postural mechanism later little pronation or further flattening of the arch can occur. Pain is slight in these cases, since but little strain can fall upon the ligaments.

Flattening of the transverse arch is most evident at the level of the metatarso-phalangeal joints, that is, at the anterior metatarsal arch. The anterior metatarsal arch is low and is maintained by the tone of the intrinsic muscles of the sole, particularly the transverse head of *adductor hallucis*, the lumbricals and the interossei. The lumbricals and interossei flex the metatarso-phalangeal joints, and in so doing raise the arch. If the normal flexion of the toes in walking is prevented by a shoe which is too short, too narrow or has an excessively high heel causing crowding of the foot into the front of the shoe, the lumbricals and interossei lose tone and the anterior metatarsal arch is deprived of its muscular support. The transverse metatarsal ligament soon stretches and the arch is persistently flattened; simultaneously, the toes become dorsiflexed at the metatarso-phalangeal joints or "retracted."

The accompanying pain, known as "metatarsalgia," in the forepart of the foot is due primarily to stretching of the ligaments and pressure on the joint capsules; but it may be also due to associated corns and callosities and to direct lateral compression of joints by a narrow shoe after the arch has flattened. One can observe the effect of the latter in the hand: when the fingers are flexed, lateral compression of the hand is painless; but if the fingers are dorsiflexed, the metacarpal arch is flattened and compression products pain in the joints. In some patients there occur paroxysmal attacks of cramp-like pain at one metatarso-phalangeal joint, usually the fourth. This affection was first described by Morton and attributed by him to compression of the plantar nerves between adjoining metatarso-phalangeal joints. Sometimes it appears to be due to overriding of the fifth on the fourth metatarsal.

Diagnosis.

Causes other than postural of depression of the arches must be first eliminated. These may be grouped as (i) congenital anomaly, (ii) paralysis of the calf or sole muscles, (iii) chronic arthritis of the tarsal or tarso-metatarsal joints, (iv) fracture at the ankle or of the tarsal or metatarsal bones. Conditions which necessitate the use of the foot in a faulty attitude, such as limitation of the range of dorsiflexion at the ankle or of the great toe, painful corns and toenails and mal-union of a fracture of the leg should be recognized. Whether the foot is flexible or rigid should be determined by voluntary and passive movement. During the examination both legs should be bare and visible to above the knees. The habitual mode of standing and walking should be observed.

Much may be learnt by inspecting the soles of the shoes. Normally, the sole is worn slightly more on the outer and back part of the heel, under the outer side of the ball of the foot and under the great toe than elsewhere. If the inner side of the heel and sole are worn down, it can be inferred that the foot is habitually pronated; if the sole under the ball of the foot shows most wear, the anterior metatarsal arch is flattened and the toes are not being used. Other features of the shoe to be noted are the length and width relative to those of each foot, the height of the heel and the width and strength of the shank.

Treatment.

The principles are (i) to remove causes of deficiency or break-down of the postural mechanism; (ii) to restore the natural form of the foot; (iii) to relieve strain of the ligaments by holding the foot in the normal attitude until the muscles have recovered their postural tone; (iv) to reeducate the postural reflex.

For foot-strain treatment consists merely of removal of the causal factors. This implies tuition in the correct use of the feet and provision of proper shoes. Both in standing and walking the feet should be parallel and pointed directly forwards. In standing, the conventional attitude with the toe pointing outwards should be corrected to the stronger position with the feet a few inches apart and parallel —the four-square position.[4] In walking, weight should be placed first on the outer side of the back part of the heel, next on to the outer border of the foot; as the heel is raised, the weight should be taken by the ball of the foot and lastly by the toes, the great toe giving the final impulse. If the foot is pointed outwards, strain is thrown on to the inner border and leverage is lessened.

In a correctly made shoe the upper fits closely around the heel and waist of the foot and is capacious enough in front to permit free movement of the toes. The heel is broad to give stability. The height of the heel is adjusted to the height of the longitudinal arch, a high-arched foot necessitating a higher heel than a low-arched foot and the height being decided by the level above the sole of a tangent to the summit of the inner border of the sole. From 1·8 to 3·75 centimetres (three-quarters to one and a half inches) is the correct height for most persons. The inner border of the sole is straight, conforming to the normal line of the great toe and the shape and width of the sole correspond to those of the foot when bearing weight. The sole is moderately thick, but is not rigid. The shank is broad and strong and is best made of leather without a metal stay which robs it of the slight flexibility it should have. The length of the shoe is as a rule two English sizes larger than the foot.

Acute foot-strain demands rest until pain and tenderness are relieved, the foot being held in the supinated position, preferably by a plaster of Paris splint and gently massaged. Treatment as for ordinary foot strain is then adopted.

In the varieties in which deformity is present, the first step in treatment is to determine whether the deformity is correctable by voluntary or passive movement or is fixed. If it is fixed, normal mobility must be regained preliminary to other treatment. Qualifications to this rule, however, exist, namely, a stiff but painless flat foot of long standing and a foot fixed by peroneal spasm. The first is not improved in function by mobilization: in the other the spasm passes off when the strained joints are relieved by correct use of the foot and a properly made shoe.

In mobilizing a rigid foot contracted ligaments are stretched and adhesions ruptured. An essential preliminary is eradication of septic foci from the body. The foot is mobilized manually or with a wrench until it is completely pliable. It is then in the state of acute foot strain. It is held in supination by a plaster of Paris splint for three weeks to allow the ligaments of the inner side to contract and then massaged and exercised. Subsequently treatment as for flexible flat foot is followed.

Limitation of dorsiflexion of the foot may be in young children correctable by exercises and passive stretching. Later it may be necessary to lengthen the *tendo Achillis;* if there is only slight limitation, it suffices to raise the heel of the shoe. Persons with this condition who complain of pain in the feet, may be advised wrongly to wear low heels, since high heels are needed. Limitation of dorsiflexion of the great toe is usually indicative of osteoarthritis of the metatarso-phalangeal joint to which treatment should be directed; if osteoarthritis can be excluded, active and passive movements to increase the range should be performed.

In treating flexible flat foot it is necessary in addition to removal of causes and instruction in the correct use of the foot, to teach exercises to reeducate the postural reflex and to alter the shoe so as to maintain the foot in the normal position while muscle tone is returning. Muscular reeducation consists of frequently repeated voluntary action to undo the deformity until the action becomes constant and habitual. To obtain this result, a set system of exercises gone through once or twice each day is not so effective as a single simple movement which the patient is directed to be always doing when the foot is both in and out of the shoe. It should be noted that in postural deformities the muscles are not weak and require no strengthening. The patient should be told to try constantly to maintain the correct position of his foot by the movement of supination—getting on to the outer border—and, if the anterior metatarsal arch is flattened, by flexing movements of his toes. He should be made to realize that his active cooperation is essential for success.

An orthopædic shoe is merely a shoe made on the lines indicated above and altered so as to counteract against deformity. For pronation deformity the shoe is so made that the foot is held supinated to just beyond the mid-position. In other words, the shoe and the foot are tilted so that the body weight is thrown definitely on to the outer border. In this way the muscles and ligaments of the inner side of the leg and foot are relieved of strain and allowed to recover tone and to shorten. A capable bootmaker can give in the making of the last the required tilt to the shoe and at the same time allow for the natural bearing points of the properly placed foot—the heel, the outer border and the heads of the first and fifth metatarsals—so that these points are depressed or "bedded" in the insole. The foot then adapts itself to the insole in the correct position. Easier, though less elegant, is the usual method of wedging the inner side of the heel and sole and placing a felt or spongy-rubber pad under the inner side of the insole beneath the longitudinal arch—known as *"valgus"* wedges and insole. The wedges are useless unless they are sufficiently high to tilt the foot on to the outer border; from four to eight millimetres (a sixth to a third of an inch) are generally required. In the Thomas method the heel is also prolonged forward on the inner side to give a strong support under the longitudinal arch. This is ugly and unnecessary in a shoe with a shank of proper width and strength.

An appliance of metal or other hard material which can be slipped from one shoe to another, is in common use and is widely sold in shops. It has the disadvantage of converting the flexible lever of the foot into a rigid system. If the sole of the shoe is weak, it is readily crushed down under a rigid appliance and the deformity not prevented; if the sole is strong and well made, the appliance is merely a hard inelastic surface which may be the cause of pain and pressure atrophy of the muscles. However, a metal plate may be valuable for severe deformity, for which also an outside leg steel and a T-strap may be required.

For flattening of the anterior metatarsal arch, the shoe is altered by fixing to the upper surface of the insole immediately behind the level of the heads of the metatarsals a "metatarsal" pad of felt or spongy rubber. This pad is a splint to the arch, holding it raised and facilitating movements of the toes. Temporary relief can be given to the metatarsal region by adhesive strapping around the forepart of the foot, applied while the arch is compressed laterally and over a pad under the sole.

References.

(1) Arthur Keith: "Menders of the Maimed," 1919.

(2) J. E. Fraser: "The Anatomy of the Human Skeleton," 1914.

(3) A. S. Blundell Bankart: "Remarks on Postural or So-called Static Deformities," *The British Medical Journal*, April 23, 1921, page 587.

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THE BLOOD IN NEPHRITIS AND URÆMIA.

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SEVERE impairment of renal function may cause such changes to occur as lead to uræmia the ætiology of which is imperfectly understood. Recent work has carried knowledge a step further towards the possible solution of the problem. E. Andrews[1] has shown that animals in which complete suppression of urine has been brought about by double nephrectomy, after remaining active for several days die suddenly without any symptoms like those of a terminal nephritis other than a high blood urea content. He points out that uræmia does not necessarily follow anuria, but that other factors such as disturbance of the acid-base equilibrium and the salt balance of the blood will cause it in the absence of normal kidney excretion. If the animals are injected with hypertonic saline solution in such quantity as to bring the chlorides of the blood to the uræmic level, they pass into a state closely resembling uræmia. Chlorides and water pass from the blood to the tissues, particularly the brain and the liver in which definite degenerative changes occur. Andrews considers that uræmic toxæmia has its cause in the products of liver breakdown and that a source of urinary protein is the liberated liver protein.

E. Andrews and Thomas[2] have shown by an application the precipitin method of Hektoen and Welker[3] that the urinary proteins in the first stages of experimental uræmia are derived from the liver and are free from blood proteins. Later, owing to renal damage caused by these foreign proteins in the blood serum proteins appear in the urine.

It is frequently found that when uræmic symptoms are first detected in a patient with renal inefficiency, the concentration of nitrogenous waste products in the blood rises much more rapidly than when the nephritis is uncomplicated by uræmia. It might be suggested that this sudden increase marks the point at which breakdown of the liver commenced to have an accelerating effect on the renal condition. Further reference will be made to this point below.

The diazo colour reaction, discovered by C. H. Andrewes[4] in the blood of patients with advanced nephritis, has offered aid in the differential diagnosis of uræmia. He found in studies of the Van den Bergh reaction that in blood from some patients with nephritis the technique of the indirect reaction gave a colour which gradually increased to a buff shade on standing twenty-four hours. If the solution was then made alkaline with sodium hydroxide, a pink colour developed instead of the expected green of azo-bilirubin in alkaline solution. Andrewes,[4] Hewitt,[5] Rabinowitch,[6] Blotner and Fitz[7] and Harrison and Hewitt[8] have investigated the reaction in serum from patients suffering from a wide variety of diseases and in none but nephritic serum has a pink colour (a sign of reaction) developed on making alkaline a mixture of an alcoholic extract of the serum and the diazo reagent. The detailed results of all these investigators show that a reaction indicates a very bad prognosis. Blotner and Fitz[7] regard the reaction as possessing much value in the differential diagnosis of uræmia and coma of non-renal origin since a reaction is obtained only in cases of advanced renal inefficiency.

In an endeavour to assess the clinical value of the diazo test, blood from patients with uræmia and with nephritis was subjected to the test and also analysed for urea, non-protein nitrogen, uric acid, creatinin and bilirubin. The last named was estimated in order to determine whether, as C. H. Andrewes[4] found in confirmation of Van den Bergh's results, a hypobilirubinæmia always occurs in nephritis and whether hyperbilirubinæmia will prevent a diazo reaction from occurring. The diazo test was controlled by applying it to blood taken from patients with coma and diseases of non-renal origin and in all these no reaction was obtained.

Bandler and Killian[9] in a consideration of the urea, uric acid and creatinin contents of the blood in nephritis decided that the relative increases in concentration of all these substances should be given weight in arriving at a prognosis. In chronic renal impairment they found a relatively greater increase in creatinin than in urea and therefore concluded that the creatinin, being endogenous while the urea is largely exogenous, gives a more dependable index of renal function. The so-called "staircase" retention of uric acid, urea and creatinin, that is, that the uric acid is the first to increase, then the urea commences to accumulate and finally the creatinin, as stated by Myers,[10] was not found by these workers in their results.

The results of the analysis of blood in the accompanying table show that in nephritis the urea, uric acid and creatinin all increase in concentration in the blood, but the relative rates of increase of these substances are not constantly maintained. If an improvement or decline in the patient's condition continues, it is seen that this is reflected in a general manner by a corresponding change in the retention of these substances. For example, in Case IV, one of chronic nephritis, between January 5, 1928, and January 17, 1928, there was sufficient improvement in the patient's condition to permit his discharge from hospital. In this period the amounts of urea, non-protein nitrogen, uric acid and creatinin were all reduced. On the patient's readmission on February 8, 1928, there was a slight increase in urea retention and a more pronounced increase in uric acid and creatinin. Nine days later urea was found in further increased amount, while uric acid and creatinin had fallen somewhat. These irregular variations continued until May 7, 1928 (when the last analysis was done), but during the whole period the clinical condition of the patient was becoming steadily worse. The conclusion of

Number of Case and Diagnosis.	Date.	Urea in Milligrammes per 100 Cubic Centimetres.	Non-Protein Nitrogen in Milligrammes per 100 Cubic Centimetres.	Uric Acid in Milligrammes per 100 Cubic Centimetres.	Creatinin in Milligrammes per 100 Cubic Centimetres.	Results of Diazo Test.	Bilirubin (Units).	Remarks.
1. Chronic nephritis and pregnancy	May 11, 1927 ..	—	79	7·2	7·4	No reaction	—	
	May 21, 1927 ..	—	—	—	—	—	—	Miscarriage occurred
	June 3, 1927 ..	109	—	6·1	7·6	No reaction	—	
	July 15, 1927 ..	123	87	7·3	7·7	No reaction	—	
2. Chronic nephritis and pregnancy	November 11, 1927	33	28	4·2	3·3	No reaction	—	
	November 28, 1927	—	—	—	—	—	—	Normal labour occurred.
3. Chronic nephritis ..	May 19, 1927 ..	42	31	4·2	2·5	No reaction	—	
	June 16, 1927 ..	33	26	3·2	2·3	No reaction	—	
	July 8, 1927 ..	23	—	3·1	2·0	No reaction	—	Patient discharged relieved.
4. Chronic nephritis ..	January 5, 1928 ..	71	54	5·0	4·2	No reaction	Not estimable	
	January 17, 1928	58	46	4·2	4·1	No reaction	Not estimable	Patient discharged relieved.
	February 2, 1928	62	46	6·5	6·3	No reaction	0·7	
	February 17, 1928	101	74	5·2	5·4	No reaction	0·8	
	February 28, 1928	70	56	5·2	8·0	No reaction	0·5	
	March 19, 1928 ..	61	46	5·9	7·5	No reaction	0·4	
	April 25, 1928 ..	—	93	5·2	5·4	Positive	Not estimable	Patient's condition becoming worse.
	May 7, 1928 ..	134	103	5·8	5·3	Positive	Not estimable	
5. Chronic nephritis ..	May 18, 1927 ..	73	51	5·8	5·0	No reaction	—	
	June 1, 1927 ..	113	83	7·1	7·3	No reaction	—	
	June 16, 1927 ..	210	196	8·3	14·3	Positive	—	Patient comatose.
	June 17, 1927 ..	—	—	—	—	—	—	Patient died in uræmia.
6. Anuria following right nephrolithotomy	July 15, 1927 ..	154	108	8·2	10·3	Positive	—	
	July 19, 1927 ..	—	—	—	—	—	—	Patient died in uræmia.
7. Chronic nephritis ..	November 28, 1927	—	—	5·7	4·9	No reaction	1·5	Patient lethargic.
	December 14, 1927	229	143	8·2	9·5	Positive	Not estimable	
	December 18, 1927	—	—	—	—	—	—	Patient died in uræmia.
8. Hydronephrosis and acute nephritis	March 19, 1928 ..	280	202	12·6	25·0	Positive	Not estimable	Uræmic symptoms apparent.
	March 21, 1928 ..	—	—	—	29·0	Positive	Not estimable	
	March 22, 1928 ..	—	—	—	—	—	—	Patient died.
9. Acute exacerbation of chronic parenchymatous nephritis	March 19, 1928 ..	226	161	9·2	24·3	Positive	Not estimable	Uræmic symptoms apparent.
	March 21, 1928 ..	—	—	—	26·0	Positive	Not estimable	
	April 2, 1928 ..	419	357	19·5	33·0	Positive	Not estimable	Patient died.
10. Acute nephritis ..	March 21, 1928 ..	133	110	7·2	7·3	No reaction	2·1	Uræmic symptoms apparent.
	March 23, 1928 ..	235	151	12·8	8·4	Positive	2·5	Patient died.

Bandler and Killian[9] that there is a relatively greater increase in creatinin than in urea in chronic renal impairment is not borne out by these results. In a few analyses in the whole series it is so, but in many analyses the creatinin retention is proportionately very much less than the urea retention. Myers's[10] statement that uric acid, urea and creatinin, in that order, tend to be retained selectively, also lacks confirmation in these results, which therefore agree with the work of Bandler and Killian[9] on this point.

In Cases VII, IX and X urea contents were found between 226 and 235 milligrammes per hundred cubic centimetres, but the creatinin contents were 9·5, 24·3 and 8·4, and the uric acid contents 8·2, 9·2 and 12·8 milligrammes respectively. The continual finding of results similar to these, showing no parallelism in the rates of increase of these nitrogenous catabolites in the blood, leads to caution in the interpretation of the results. In prognosis one should be guided only by a definite change in the concentration of any one of these substances and therefore information of greater value will be obtained if estimations of all three, rather than of only one, are done. Urea and non-protein nitrogen have similar rates of increase and which of these is estimated is immaterial.

In Cases VIII, IX and X it is seen in the table that the renal function rapidly became worse after the onset of uræmia. If E. Andrews's[1] [2] line of reasoning to explain uræmic toxæmia is followed, it would appear that the products of liver breakdown which commenced at the time of onset of uræmia, had caused such further damage to kidneys, already impaired, that their power to excrete nitrogenous waste products was reduced to a very low level indeed. In these circumstances the salt balance of the blood would become still further disturbed and liver degeneration would be hastened. A vicious circle of malfunction would be established. To the acidosis due to the derangement of the buffer salts of the blood would be added the toxæmia resulting from liver decomposition products. While further evidence on the question is, of course, desired, it is seen that in this direction there may be at least a partial elucidation of the ætiology of uræmia.

In seven of the ten cases quoted in the table the blood yielded a positive response to the diazo test. In six cases death occurred within short periods after the first reaction was found. In Case IV a reaction was first obtained on April 25, 1928, and the patient is still alive, but his condition clinically is bad.[1] With a blood creatinin of 5·3 milligrammes per hundred cubic centimetres and a blood urea of 134 the result of the test was positive, whilst in other cases, for example, Case X, with a blood urea

[1] Since this paper has been sent to press the patient has died. This case, therefore, illustrates the prognostic value of a diazo reaction when the retention of nitrogenous waste products has not reached a very high level.

of 133 and creatinin 7·3 no reaction was obtained and a reaction was obtained when the creatinin had risen slightly to 8·4 and the urea had largely increased to 235. A reaction then is sometimes obtained in blood in which the retention of nitrogenous catabolites is not very great, or it may appear when only a small increase has occurred in the retention of some of them. Unlike the urea, uric acid and creatinin contents of the blood, the diazo reaction is not interpreted quantitatively and the mere occurrence of a reaction of any intensity of colour is of grave import and indicates a bad prognosis. In Cases VIII and IX in which the blood creatinin rose to the very high figures of 29 and 33 respectively just before death, the pink colour of the diazo reaction was very deep, but in Case IV with creatinin 5·3 the intensity of the colour was much less. In other cases colours of intermediate intensity between these two extremes occurred. The intensity of the pink colour varies approximately with the degree of retention of urea, uric acid and creatinin, but the significance to be attached to a positive result is the same in all cases.

The occurrence in normal urine of a substance giving a diazo reaction has been demonstrated by fractionating it from the urinary pigment urochrome. The method used is described in detail below. The reaction obtained from the final alcoholic fraction of urine which represented a two-thirds concentration of the original urine, gave a deep pink colour which faded very slowly in the same manner as a strong reaction from serum. One concludes that the same substance is responsible for the reaction in each case and that a reaction from serum indicates that a substance, very readily excreted by normal kidneys, is being retained by kidneys with greatly impaired function.

Although a hypobilirubinæmia is shown in the table not to occur invariably in nephritis, it occurred in all cases in which the result of the diazo test was positive except Case X. In that patient a reaction and hyperbilirubinæmia occurred together, the increase of bilirubin in the blood being due to secondary carcinoma of the liver. Despite the green colour of azo-bilirubin in alkaline solution the pink of the diazo reaction was readily distinguished.

The utility of the diazo reaction in the differential diagnosis of uræmia has been referred to by Rabinowitch[6] and Blotner and Fitz.[7] Their conclusions have been confirmed in the present work. In cases of coma a reaction was found in the blood serum only when the coma was uræmic and then it occurred invariably. Specimens of serum from patients with coma of non-renal origin, arteriosclerosis and other diseases all gave no reaction. Being easily and rapidly performed, the test may be regarded as a useful aid in the differential diagnosis of uræmic coma, a positive result indicating uræmia and absence of reaction definitely excluding it.

Experimental Details.

The method of carrying out the diazo reaction that has been adopted is as follows:

Diazo reagent (as for the Van den Bergh test):

Solution *A*: Sulphanilic acid 1 gramme, hydrochloric acid, concentrated, 15 cubic centimetres, distilled water to 1,000 cubic centimetres.

Solution *B*: Sodium nitrite 0·5 gramme, distilled water to 100 cubic centimetres.

Mix 25 cubic centimetres of solution *A* with 0·75 cubic centimetre of solution *B*. The reagent thus prepared remains good for several days.

To one cubic centimetre of serum (or oxalate plasma) add two cubic centimetres of aboslute or 95% alcohol and shake well. Centrifuge or filter. To one cubic centimetre of the alcoholic extract add 0·5 cubic centimetre of alcohol and 0·25 cubic centimetre of the diazo reagent. Boil for half a minute. A buff colour developes if the result is going to be positive. Add 10% sodium hydroxide, drop by drop, with shaking. A reaction is indicated by a bright pink colour, usually appearing with the first drop of alkali and persisting for a variable time.

Urea estimations were done in part by Maclean's method[11] and in part by Myers's method[12] and an alcoholic solution of urease was used. The micro-Kjeldahl digestion for the estimation of non-protein nitrogen was done according to the method of Myers.[12] Creatinin was estimated by Myers's technique,[12] special care having been taken in preliminary purifications of the reagents used. For the estimation of uric acid the Folin and Trimble[13] modification of Folin's original method was followed. Experience of the methods for creatinin and uric acid showed that careful attention to technique and choice of pure chemicals rendered them quite satisfactory.

The procedure followed in the examination of normal urine for a substance giving the diazo reaction was as follows: Three hundred cubic centimetres of urine were concentrated *in vacuo* at 40° to 45° C. to a paste. This was rubbed up in a mortar with four successive ten cubic centimetre quantities of absolute alcohol. The alcoholic extracts were centrifuged and the supernatent fluids combined and concentrated to a very small volume (less than one cubic centimetre). The resulting viscous liquid was poured with constant stirring into two hundred cubic centimetres of absolute alcohol which was then filtered. The filtrate, lightly coloured with urinary pigments, gave a strong diazo reaction. The first alcoholic extract of the paste of concentrated urine failed to give a definitely positive result, although a rapidly fading pink colour was observed in the highly coloured brown solution.

Summary.

1. The interpretation of the urea, uric acid and creatinin contents of the blood in nephritis has been discussed. The advantage of estimating all these substances has been pointed out.

2. The rapid increase in retention in the blood of nitrogenous catabolites after the onset of uræmia has been discussed in the light of recent work on experimental uræmia.

3. The diazo reaction in nephritic sera has been shown to be of value in prognosis. Its utility as an aid in the differential diagnosis of uræmia has been mentioned.

4. The diazo reaction has been shown to occur independently of the bilirubin content of the blood.

5. A substance giving a reaction resembling in all respects a positive diazo reaction in serum has been found to occur in normal urine.

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Reviews.

A NEW HANDBOOK ON PSYCHIATRY.

"MENTAL DISORDERS," by Dr. Hubert J. Norman, is a new arrival in the field of handbooks on psychiatry for students and practitioners.[1] A very laudable attempt has been made to cover the whole ground, as is shown by the devotion of chapters to such matters as borderland or eccentric types and sexual perversions, an historical survey, Mowgli types and wild children and examples in life and literature. The latter contains a useful bibliography which betrays wide and varied reading on the part of the author who uses quotations freely to embellish the text.

Dr. Norman is strictly orthodox in his views not, as he states, because it is necessary to "pooh-pooh whatever is fresh and new," but because much of modern psychiatry is in an inchoate condition. As his book deals chiefly with the major psychoses and psychoneuroses—*dementia præcox*, delusional insanity, stupor, melancholia, mania *et cetera*—a somewhat scanty reference to the psychological factor is not therefore of such importance. For the same reason the somewhat pessimistic outlook as to treatment is understandable. However, his remark concerning *dementia præcox*, that "it is better in any case to adopt a wise empiricism than to do nothing," is worth quoting as an example of his viewpoint. In that disease he makes favourable comment on the malarial treatment. In epilepsy the omission of the use of borax in combination with bromides or of cerebro-spinal puncture in *status epilepticus* ought to be rectified in a subsequent edition. More might have been written of endocrine disorders, particularly concerning the possibility of mixed thyreoid states. Classical myxœdema and cretinism as described are rarities.

The chapter on the association of mental disorder with the epochs of life contains some suggestive paragraphs and the author has a word to say about intensive education which assumes that there is a "mind" which can be stuffed and not, as is actually the case, a long-suffering nervous system which has to be developed. Of adolescence he remarks that "'as the twig is bent, the bough is inclined' is applicable to the human being . . . when the storm comes a crash will take place. Possibly the worst one is *dementia præcox*." The author dismisses the subject of dreams rather summarily, preferring a theory which would account for Ingoldsby's dream—"It's that confounded cucumber I ate and can't digest"—rather than accept any Freudian interpretation. In justice to the psycho-analytical school, it may be stated that its findings are worthy of more attention than Dr. Norman bestows.

Contrary to the usual method, chapters on psychology are found towards the end of the book. They are concise and can be easily assimilated by the tiro in this branch of medicine. The section on general treatment is written from the point of view of the medical superintendent of an institution and is obviously the fruit of long experience. The idea that contact with other mental patients is injurious is strongly combated. Practical hints on tube feeding, care of the bowels and bladder and insomnia are important. An observation concerning the difficult problem of suicide is full of wisdom, namely, "that the most anxious periods are the earlier stages of the attack when the patient has more initiative and the later ones when, although depression remains, initiative is regained."

For the rest, the book is compact, well printed and has fifty-seven appropriate illustrations. The terminology is simple and understandable. The reader will pay the author the compliment of wishing that the work was fuller, but in a handbook this is a necessary evil. A pint pot can hardly be expected to contain more than a pint.

[1] "Mental Disorders: A Handbook for Students and Practitioners," by Hubert J. Norman, M.B., Ch.B. (Edinburgh), D.P.H. (Edinburgh and Glasgow); 1928. London: E. and S. Livingstone. Crown 8vo., pp. 478, with illustrations. Price: 14s. net.

A BOOK FOR MOTHERS.

"ADVICE TO THE EXPECTANT MOTHER ON THE CARE OF HER HEALTH" is the title of a very practical book written by Dr. F. J. Browne, Professor of Obstetrics in the University of London.[1] In the space of thirty-eight pages there has been given sound advice in simple language of the benefits of antenatal care, normal pregnancy, hygiene of pregnancy, common disorders and their treatment, preparations for confinement and hints on breast feeding and the care of the baby.

It is noteworthy that the risks of marital relations during the last three months of pregnancy as well as of those following self examination are emphasized. In view of the importance of postnatal examination the value of the book would have been increased by a chapter on the care of the mother during and after the puerperium.

Advice along the lines of this book is needed by all expectant mothers and it can be recommended to practitioners seeking literature well written and at a reasonable price for their patients. A good index has been included.

[1] "Advice to the Expectant Mother on the Care of her Health," by F. J. Browne, M.D., D.Sc., F.R.C.S.E.; Second Edition; 1928. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 48. Price: 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 9, 1928.

Teaching of Obstetrics in Melbourne.

ON May 18, 1928, the Premier of Victoria announced that the Government had agreed to cooperate with the University of Melbourne in the establishment of a chair of obstetrics. He stated that the necessary money would be provided by Parliament as soon as the arrangement could be made.

It may be useful to recall the events which have occurred in Melbourne during the past few years in regard to the teaching of obstetrics. In August, 1925, the Council of the University of Melbourne announced that the Trustees of the Edward Wilson (*The Argus*) Trust had given the sum of £10,000 for the purpose of endowing research in the science of obstetrics. The Council had appointed a committee to advise the University on the best method of carrying out the wishes of the Trustees. The committee comprised Professor R. J. A. Berry, Dr. R. H. Morrison, Dr. H. C. Lloyd, Dr. C. H. Kellaway, Dr. S. S. Argyle, Sir George Syme, Dr. Felix Meyer, Dr. R. H. Fetherston and Dr. J. W. Dunbar Hooper. It was decided to appoint a director of obstetrical research, who would be required to make a survey of obstetric work in Victoria and to carry out investigations with the object of ascertaining the causes of maternal mortality and morbidity and of suggesting methods for prevention and treatment. The appointment was to be for a period of two years at a salary of £2,500 a year. In September, 1925, Dr. R. Marshall Allan, M.C., was appointed and entered upon his duties within a short time. A year later a verbal promise was given by the Trustees of the Edward Wilson (*The Argus*) Trust that the sum of £60,000 would be offered to the University at the end of the term of office of the Director of Obstetrical Research for the purpose of founding a chair of obstetrics to be known as the Edward Wilson chair, in memory of the founder of the Trust. The Government of Victoria was approached and a tentative arrangement was formulated according to which the Government would provide sufficient funds for the establishment of a chair of medicine simultaneously with the chair of obstetrics. The work of the Director of Obstetrical Research is well known to the medical profession throughout the Commonwealth. His interim report was published in this journal on January 1, 1927, and his final report has occupied practically the whole of last week's issue. It is unnecessary to emphasize the fact that the selection of the committee has been amply justified from every point of view. At the end of two years an extension of time for a period of six months was granted, in order to permit Dr. Marshall Allan to complete the work he was carrying out. At this time the Government had taken no steps to carry out its part of the undertaking. The Trustees learned that as various plans had been under consideration for the development of the University of Melbourne and had not come to fruition, the Government was not prepared to establish a chair of medicine for the time being. They therefore withdrew their offer to place the sum of £60,000 at the disposal of the Council of the University for a chair of obstetrics. It appears that the management of the Bush Nursing Association obtained the ear of the Trustees and drew their attention to a report that had been drawn up by Dr. Marshall Allan on the records for the year 1927. In his report he referred to the fact that the nurses belonging to this organization had attended 2,273 women in their confinements between 1922 and 1927, without a single death. He attributed this excellent result to the continuous antenatal care given by the medical practitioners in charge, to the removal of all women with signs of complications to properly equipped hospitals and to the cooperation of well-trained nurses with the medical practitioners. These facts have been quoted incompletely and it has been made to appear that the saving of life has been the result of the bush nurses' work. The maternal mortality in Victoria from 1922 to 1926 has increased from 3·36‰ to 5·84‰, so that the number of lives saved may be estimated at eight. This is, of course, eminently satisfactory,

but it is unwise to exaggerate its significance. There has been much open talk in Melbourne concerning a proposal that the £60,000 originally intended for the chair of obstetrics, is to be handed to the Bush Nursing Association for development of its work particularly in the Mallee district. Dr. Marshall Allan has written strongly in appreciation of this organization and it will be admitted by all interested persons that it is serving a very useful purpose by providing well-trained nurses for the people of isolated districts. On the other hand, there is another aspect of the movement which is not admirable. Bush nurses are often placed in districts where there are no medical practitioners and at times these nurses undertake what is in effect medical practice. That a nurse, even if she be highly trained and competent as a nurse, is not qualified to diagnose and treat illness and injury, must be admitted by everyone. The *Medical Acts* exist to protect the public from the dangers of irresponsible medical practice by unregistered persons and it would be disastrous if encouragement were given to any organization to develope unqualified practice on the pretext that there are outback districts in all the States where a medical practitioner is not easily obtainable.

Fortunately the rumour that the Trust money is to be given to the Bush Nursing Association is still unconfirmed. It is therefore possible that wiser counsels may prevail and that the Trustees will yet honour the promise given but a short time ago. The offer of the Government of funds to provide for a chair is unlikely to meet with approval. It will be noted that the original proposals of the Trustees of the Edward Wilson (*The Argus*) Trust was that the sum of £60,000 would be allocated for the purpose and that the chair was to form the corollary of the directorship of obstetrical research. The Director received a salary of £2,500 a year. If the teaching of obstetrics is to be raised to a high level of efficiency, it is essential that the professor should be a whole-time officer and not be allowed private practice. He would hold an honorary position in one of the teaching hospitals and might be allowed to meet his colleagues in consultation, provided that this did not interfere with his organizing and teaching duties. He must be an obstetrician of repute and it therefore follows that a reasonable salary must be offered. The Trustees have indicated the amount of salary that should be paid, both when they instituted the directorship of research and when they announced that £60,000 would be given for the chair. The Council of the University will do well if it recognizes the present opportunity to improve its medical school and to set an adequate standard in the teaching of so important a subject as obstetrics. The lives of the mothers of the community are in the hands of the medical profession and it is of the utmost urgency that the members of the medical profession should be trained to be skilled and safe obstetricians in the modern sense. The problem of the dangers of child-birth is œcumenical; it is becoming more urgent every day in every country. It is to be hoped that the Trustees of the Edward Wilson (*The Argus*) Trust will recognize that their generosity has resulted in a most admirable beginning and that their continued generosity along the same lines will place the name of Edward Wilson in the front rank of the benefactors of mankind.

Current Comment.

CHOLECYSTOGRAPHY.

No one will deny that the introduction by Graham of the use of sodium tetraiodo-phenolphthalein in suspected disease of the gall bladder has been the means of placing the treatment of gall bladder disease, more particularly from the surgical aspect, on a much sounder basis. In September, 1927, the subject was discussed in these columns from the point of view of reliability in connexion with a paper by Anderson Stuart, published in the same issue. No mention was made of the possibility of untoward results as far as the patient is concerned. In 1926 Huddy recorded the death of a patient who had received a dose of 5·5 grammes of sodium tetraiodo-phenolphthalein. Graham attributed this patient's death to the size of the dose. He stated that he never gave more than 0·06 gramme per kilogram of body weight. Disabilities of lesser degree, however, have been reported. In a series of cases reported by L. R. Whitaker 68% of patients had no reaction, 8% had nausea, 12% had nausea and vomiting and 12% had diarrhœa. In no instance were the symptoms alarming. Whitaker's patients received the drug by mouth. Graham reported that in a series of 205 patients who received the drug by intravenous injection, 53% had reactions, in 36% the reaction was

mild, in 17% it was less mild and in none was it alarming.

B. M. Dick and V. G. H. Wallace have recently reported several cases in which the results were so disastrous that two patients died and they have given details of experimental work carried out by them on the toxic effects of the dyes.[1] The first patient was a robust miner, aged forty years. He had signs and symptoms of gall bladder disease and an intravenous injection of 5·5 grammes of sodium tetraiodo-phenolphthalein was given. The patient became worse three and a half hours after the injection and he died of acute hæmorrhagic pancreatitis. The second patient was a woman, aged fifty years, who suffered from dyspepsia regarded as being due to a lesion of the gall bladder. She received 3·5 grammes of the same drug as the previous patient. She subsequently became severely jaundiced, operation was performed and two gall stones were removed. Recovery was uneventful. The third patient was an unmarried girl, aged twenty years, who was regarded as suffering from gall stones. She was deeply jaundiced and tenderness was present in the upper part of the abdomen. She received five grammes of the same salt as the other two patients, subsequently became acutely ill and died thirty hours after taking the drug. At *post mortem* examination appearances suggestive of acute yellow atrophy of the liver were found. The authors point out that the first patient received a dose larger than that which is usually given by Graham; it was the dose which it was customary to use at the particular hospital at that time. It was in their opinion difficult to decide whether the third patient suffered primarily from acute yellow atrophy or from simple catarrhal jaundice. The patient did not appear very ill and there was no contraindication to the use of the drug. It was regarded as certain, however, that the administration accelerated the course of the patient's illness and contributed to its fatal ending.

Dick and Wallace have carried out some experimental observations on cats and rabbits. Cats were selected because of the resemblance of their biliary system to that of the human being. A dosage of 0·05 gramme of sodium tetraiodo-phenolphthalein per kilogram of body weight of the animal was employed. In the first place it was found that when the common bile duct was ligatured, the pancreatic juice always contained small but appreciable amounts of the drug. In the second place it was found that when large doses were brought to the pancreas either by the inferior pancreatico-duodenal artery or by the pancreatic duct, hæmorrhagic and necrotic changes resulted. In the third place it was found that in the rabbit and cat no changes were detected in the pancreas after a single injection. In nine rabbits the common bile duct was ligated before injection. Four of the animals died of acute hæmorrhagic pancreatitis, in two swelling with some fat necrosis was present and no changes were seen in the remaining three rabbits.

[1] *The British Journal of Surgery*, January, 1928.

In the next series of experiments the effect on the pancreas of bile containing the sodium salt was investigated in cats. The bile was injected into the pancreatic duct. Details of the procedure need not be given, but in five cats treated in this way acute pancreatitis with fat necrosis resulted in death. As a control the pancreatic ducts were perfused with saline solution containing an amount of the drug equal to that present in 0·5 cubic centimetre of bile from the gall bladder after an intravenous injection of the drug. The normal acinous structure of the gland was almost totally lost. In places whole blocks of acini were seen undergoing necrosis. There were no capillary hæmorrhages. When normal saline solution only was injected, no pathological changes occurred. From this it is inferred that when stones are present in the common bile duct in cholelithiasis and the conditions are otherwise favourable for the retrojection of bile into the pancreas, the danger of the occurrence of acute pancreatitis will be much greater if the regurgitated bile contains the phenolphthalein salt. Experiments were undertaken which showed that the drug has a greater toxicity upon the liver in the presence of obstructive jaundice. In regard to the kidneys the changes observed were never more than slight when diagnostic doses were employed. In a number of cases the glomeruli were swollen and the epithelium of the convoluted tubules manifested an early stage of cloudy swelling; the cells of the collecting tubules were unaffected. After the biliary system had been excluded, the drug was eliminated almost entirely by the kidneys. The rate of excretion was slow; the agent was detected in the urine as long as eighteen days after the intravenous injection.

The first thing to be remembered in connexion with this work is that the conditions under which many of the experiments were carried out do not exist in the human body. At the same time they show that the drug in question is a potential source of danger. The dose in the case of both patients who died, was higher than that recommended by Graham and the warning may well be taken to heart. At the same time it is not unlikely that the organs affected were already the seat of early pathological change. In some instances it will undoubtedly be difficult to exclude lesions of this description. The symptoms reported by workers such as Whitaker and Graham, to which reference has already been made, are certainly due to the action of the drug and are in all probability accompanied by some pathological change, although of the smallest degree. It is true that Graham claims to obtain more satisfactory results with sodium phenol-tetraiodophthalein, an isomer of sodium tetraiodo-phenolphthalein and that Carman advocates the use of tetrabrom-phenolphthalein by mouth and of tetraiodo-phenolphthalein when there is some doubt about the final result. Nevertheless caution must be practised by all who use the test. It were better sometimes to be in doubt as to the diagnosis than to risk inflicting permanent damage on cells which are already putting up a fight to maintain their existence.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Alkalis in Scarlet Fever.

B. A. PETERS (*The Lancet*, December 10, 1927) records his study of the effect of alkali treatment in diphtheria and scarlet fever. He has previously shown that an acidosis with ketonuria occurs in toxic forms of diphtheria and that sodium bicarbonate in doses of two grammes (thirty grains) every four hours prevents pernicious vomiting and circulatory failure. In the comparison of figures of those who developed the cardiac vomiting syndrome and were treated with alkali, with those who were not so treated, it was found that only 1% of the former died, whereas deaths among the latter amounted to 2%. These figures were taken during different years and during epidemics of varying severity; ten thousand cases were studied in all. The alkalis were given until the urine became alkaline to litmus paper. This took three or four days. Alkalis relieved severe vomiting in addition to lessening the incidence of death. In scarlet fever alkalis were given during the acute stage for about a week as above. Osman and Berry claimed that alkalis given in this way prevent nephritis and possibly rheumatism and endocarditis. Peters analysed eight thousand cases of scarlet fever and found no difference in the frequency of these complications in those treated with and without alkalis.

Uses of Ephedrin.

EPHEDRIN, the alkaloid derived from the plant *Ephedra vulgaris*, was rediscovered by K. K. Chen in 1923, but T. L. Althausen and I. C. Schumacher (*Archives of Internal Medicine*, *December* 15, 1927) point out that a decoction of the plant has been used in China for more than five thousand years as a diaphoretic and antipyretic. The authors employed the drug in the treatment of nine patients suffering from vascular hypotension, in doses of fifty milligrammes thrice daily; the drug was given *per os*. In every instance a rise of blood pressure was noted from five minutes to one hour after the dose. The rise of pressure varied between nine and forty millimetres and continued for periods ranging up to four hours. Doubtful benefits follow the use of ephedrin in the treatment of severe hæmorrhages. Favourable results have been reported following its administration in toxic erythema, but no improvement seems to occur in severe urticaria. In the treatment of asthma, the reports are more favourable, 56% of asthmatic patients having been completely relieved and 24% partially relieved by its use. In all the patients treated for the relief of asthmatic symptoms, the vital capacity was estimated and blood pressure readings taken before and after the administration in order to eliminate the purely psychical effect of the drug. The vital capacity was found to be increased by as much as 35% and the systolic blood pressure on an average rose twenty millimetres. These findings were accompanied by definite subjective improvement, leading to absolute freedom from asthmatic symptoms. Undesirable effects (nausea, vomiting) may arise if injections of adrenalin precede the use of ephedrin. As to the possible evils attending the use of the drug, the chance of renal damage must be remembered, since red blood cells and casts were found in the urine of several patients after they had taken ephedrin.

Treatment of the Cardio-vascular Lesions of Syphilis.

MARCEL PINARD (*La Presse Médicale*, January 21, 1928) states that for the long-continued treatment of the cardio-vascular manifestations of syphilis small doses of mercury are to be preferred to small and ineffective doses of "Arsenobenzol." He advocates the employment of massive doses of "Novarsenobillon" and cites the history of a patient with an aortic aneurysm who improved rapidly after two doses of 1·2 grammes of the drug and who continued to benefit by this heroic treatment. To a second man with an aneurysmal dilatation of the external carotid artery, first observed in the year 1922, Pinard administered in all 150 grammes of "Novarsenobillon" during six years; during this period the patient continued to follow his occupation. Pinard deprecates the cautious methods adopted by many clinicians and considers that only by the use of large dosages are patients able to enjoy anything resembling normal health and activity.

Dextrose in Obesity.

B. GORDON AND E. STANLEY (*The American Journal of the Medical Sciences*, January, 1928) report some results of treatment of obesity by the use of dextrose. Obese student nurses were given a diet consisting of one or two pieces of bread twice daily, one hundred grammes of meat, clear soup, one small potato, butter, milk and fruit if desired, with any quantity of 3% or 5% vegetables. The nurses were instructed to walk twelve to twenty "blocks" each day and during the time they were taking exercise, to eat one hundred to one hundred and seventy-five grammes of dextrose candy in small amounts at a time. This diet was continued for three weeks, then discontinued and a restricted diet of 1,200 to 1,400 calories allowed during two weeks without dextrose. After this the diet with dextrose was resumed and this was again followed by a non-dextrose diet and so on. In three months the majority of the nurses treated lost from five to seven kilograms without any untoward symptoms. This treatment was based on the idea that dextrose would be quickly absorbed, especially during exercise, and that it would not tend to the laying down of fat. Dextrose candy consisted of one thousand grammes dextrose and three hundred grammes of honey made up into toffee.

Treatment of Malaria.

G. R. ROSS (*The Journal of Tropical Medicine and Hygiene*, October 15, 1927) records the results of treatment of malignant tertian malaria by other means than by quinine. Quinine is contraindicated when idiosyncrasy or hæmoglobinuria exists. Arsenical preparations have been useful in benign tertian and quartan ague, but neither these nor methylene blue are effective when *Plasmodium foliciparum* has been the infecting agent. "Mercurochrome 220 soluble" has been used in malaria, usually without much success. Ross injected four or five cubic centimetres of 1% mercurochrome intravenously in several cases of malaria. The dose was repeated several times every second or third day. Good results were obtained temporarily in five out of six cases. "Plasmochin," a synthetic quinoline derivative, was also tested in malignant malaria, 0·02 gramme being given five times daily for five days, omitted for four days and repeated for three days. This sequence was followed until seventeen days' treatment had been given. Two out of four patients relapsed after obtaining temporary benefit.

Asthma and Bronchitis.

E. J. KUH (*The Medical Journal and Record*, November 16, 1927) describes a method of treatment of asthma and bronchitis by spraying. A de Vilbiss atomizer, Number 56, is used; the curved tip of this is introduced behind the tongue and the patient inhales slowly with half-parted lips. In correct inhalation all spray disappears from the mouth. The atomizer emits a very fine spray. The composition of the solution used is: menthol 1%, creosote 1%, camphor 0·5%, oil of pine needles 2% in approximately thirty cubic centimetres of alboleine or similar mineral oil. Thirty cubic centimetres should be inhaled at each treatment. Asthma, acute and chronic bronchitis, whooping cough and hay fever are benefited by this treatment. In hay fever the patient closes the mouth and breathes in and out through the nose.

Migraine.

C. L. HARTSOCK (*The Journal of the American Medical Association*, October 29, 1927) refers to the treatment of migraine. He regards the condition as evidence of stasis and possibly toxin formation in the duodenum and considers that dilatation of the duodenum is not rare in migraine. He advocates restriction of carbohydrates and of eggs and milk. No white bread, wheat cereals, potatoes, eggs or milk are allowed. Frequent feeds and high fat diet are advised. Exercise and abdominal supports are to be used when visceroptosis is pronounced. Exercise is necessary in all cases. Calomel and a morning saline are given once a month and in some cases a morning saline once a week. Rest in bed for

ten hours a day and "Luminal" or bromides are given at first. Duodenal drainage is used with success in some instances.

Posterior Pituitary Extract.

S. L. GARGLE, D. R. GILLIGAN AND H. L. BLUMGART (*New England Journal of Medicine*, March 15, 1928) have sought to determine whether the pressor or the oxytocic principle is responsible for the well known antidiuretic effect produced by injection of extract of the posterior part of the pituitary gland. Four normal subjects were given a constant salt and protein diet and the fluid intake and urinary output were measured. After twenty-four hours on this diet, one litre of water was given at 7 a.m. and the urine was collected every hour for seven hours, basal conditions being observed by the omission of breakfast and by luncheon being delayed until 2 p.m.. On the next day 0·25 cubic centimetres of "Vasopressin" was injected hypodermically and a litre of water given as before at 7 a.m. On the third day the same procedure was observed, but oxytocin was substituted for the "Vasopressin." In these four normal men the injection of a single dose of "Vasopressin" checked the excretion of a litre of water for five or six hours. Injection of oxytocin had little or no effect. In two patients with *diabetes insipidus* the "Vasopressin" effectively controlled the thirst and polyuria, but oxytocin again proved ineffective. It was also found that intranasal administration of "Vasopressin" on cotton wool had a powerful antidiuretic effect.

NEUROLOGY.

Disorders of Sensation Produced by Cortical Lesions.

GORDON HOLMES (*Brain*, October, 1927) who originally collaborated with Head on the subject of disorders of sensation produced by cortical lesions writes that since then he has had the opportunity of examining sensation in very large numbers of men with gunshot wounds of the head both during and after the war and in other instances and finds that his independent investigations agree closely in result with Head's latest conclusions. The qualities of sensation localizable in the cerebral cortex are: (i) The appreciation of relationships in space, (ii) the faculty of reacting appropriately to tactile stimuli of different intensity and (iii) the recognition of similarity and difference of test objects of various weights and sizes. Pain and thermal sensibility and the vibration sense are not altered by disease confined to the cortex. As to the distribution of sensory disturbance of cortical origin this is always limited to the opposite side of the body. Usually it is more pronounced in the distal than in the proximal segments of the limbs, probably owing to the higher and more complex sensory functions of the hands and feet than those of the arms and thighs and to their wider representation in the cortex. Moreover, it frequently happens that the disturbance of sensation is apparently limited to or at least more pronounced on one side of a limb, as on the radial or ulnar border of the hand or on the inner or outer aspect of the leg. This notwithstanding, the terms "axial" and "radicular" are incorrect and misleading when applied to such disturbances. Numerous observations, however, point to a topographical representation of the fingers in the sensory cortex, in a way similar to the motor representation in the precentral gyrus. As to the distribution of the sensory zone of the cerebral cortex most of the older physiologists and some clinicians, including Head, insisted that sensation might be disturbed by lesions limited to the precentral gyrus. But Collier's own observations have led him to conclude that the sensory area lies entirely behind the central fissure and that the precentral gyrus has no sensory functions. The posterior limits of the area are less easy to define; it certainly extends over the whole of the postcentral gyrus, over the greater part at least of the superior parietal lobule and probably on to the anterior part of the supramarginal gyrus. Corresponding with the focal representation in the motor area, there is in the postcentral gyrus the head segment, fingers, arm, trunk, leg and foot represented in this order from below upwards. It is also probable that special sensory functions are separately represented in the cortex. On this point Collier's conclusion is that the identification of form, the discrimination of compass points and perhaps the comparison of weights are more widely represented in the cortex than tactile sensibility or the appreciation of position or movement. The sense of position suffers chiefly when the postcentral gyrus is involved; more complex faculties, as the recognition of form, have a wider distribution, while altered responses to tactile stimuli and loss of localization are found chiefly when the lesion lies immediately behind the central fissure.

Epileptic Variants.

S. A. KINNIER WILSON (*Journal of Neurology and Psychopathology*, January, 1928) relates his observations of a large number of patients whose conditions present intimate or remote similarities to the phenomena of epilepsy, but which nevertheless do not seem to resemble each other, at least not superficially. He calls these epileptic variants and classifies and describes them as follows: (1) Motor variants. (*a*) Myoclonic or regional epilepsy. This common variant takes the shape of irregular twitches of a limb in multimuscular groups and in colloquial parlance is often termed "the jumps." In view of the indifference of the average patient to this condition until or unless major fits appear, its early diagnostic significance is important. (*b*) *Epilepsia partialis continua.* Here the twitching is confined to one segment of the body, nearly always a peripheral part such as the wrist or fingers and is practically continuous between ordinary major fits. It suggests but is not associated with organic lesion. (*c*) Tonic epilepsy. These are fits characterized by tonic contractions only, to the exclusion of the clonic element. Such fits usually have an organic basis. (*d*) Coordinated epilepsy. This term denotes those cases wherein the movements seen during the attack are coordinated and seemingly purposive. They may occur before, during or after the convulsive phase. Fugues would come within this category. (*e*) Inhibitory or akinetic epilepsy. This refers to attacks of immobility in which the patient states that his arm "falls dead." Trance, catalepsy, narcolepsy and cataplexy may be inhibitory epilepsy. (2) Sensory variants. (*a*) Reflex epilepsy. This is present when an epileptic seizure follows an extrinsic sensory excitation, for instance a loud sound; it is also illustrated by the little boy who had a fit whenever he was undressed. (*b*) Sensory epilepsy. In such condition the aura and march are purely sensory. (*c*) Affective epilepsy. This denotes fits consecutive to extrinsic or intrinsic affective stimuli—fits produced by emotion. They were recognized long before the war reproduced them in abundance. (3) Psychical variants. The bad temper of the epileptic is an example. (4) Visceral variants. These are represented by vaso-vagal attacks, gastric or epigastric and cardiac sensations, respiratory and vasomotor symptoms, and other visceral disturbances.

Shyness.

F. A. HAMPTON (*Journal of Neurology and Psychopathology*, October, 1927) points out that shyness has recently been drawn into the compass of clinical psychology by some who suggest that schizophrenia is essentially a chronic and exaggerated degree of what in normal people is called embarrassment or "morbid self-consciousness." There is this difference, however; in schizophrenia a morbidly active instinct is ever present and dominates the personality, in shyness it is only intermittent and the balance of the personality is, therefore, not greatly disturbed. The conflict in shyness lies between an "urge" to reach upwards to the normal level from a position of imagined inferiority and a deterrent fear of failure. The actual inferiority is never one that the shy person admits freely to himself or of which he is fully aware. Some of the commoner forms of inferiority are sense of cowardice, inability to play games, physical unattractiveness and social inferiority. Most of the phenomena and reactions of shyness can be traced to fear. For some reason the world is tolerant of shy persons, accordingly shyness has its advantages. In the treatment of shyness there is no magic formula which will impart the desired self-confidence, but it may help to disclose to the patient the source of his feeling of inferiority.

Special Abstract.

HOUSING CONDITIONS AND MEASLES.

A REPORT has been issued by the Medical Research Council of the Privy Council of an inquiry made by Dr. James L. Halliday into the relationship between housing conditions and the incidence and fatality of measles.[1] Dr. Halliday is an officer of the Public Health Department in Glasgow. After a brief introduction in which reference is made to outbreaks of measles in the South Sea Islands, in Willesden, England, and in Paris, Dr. Halliday passes on to his own investigation. He states that the available data obtained by notification, even in districts where measles is compulsorily notifiable, is of necessity incomplete. In a large number of instances no medical practitioner is called to see the patient. The data must be even more incomplete in a district, such as Glasgow, where notification is mainly attained through the sanitary inspectors and the school attendance officers of the education authority. When a large epidemic is in progress, it is so overwhelming that sanitary inspectors cannot possibly trace every infection. There is also reason to believe that during the time of epidemic every case of measles occurring among children attending school, is not notified to the Public Health Department. The crude record of the incidence of measles obtained by notification cannot be used as far as the various sections of the community are concerned. It was for these reasons that during the epidemic of measles in 1925 to 1926 a special investigation was made in Glasgow into the incidence of measles in selected tenement areas, in housing scheme areas and in schools with a view to determining the influence of the different environments.

It is necessary to point out that the term "tenement" has a different significance in Scotland and in England. In Scotland the word tenement signifies a block or building consisting of several storeys on each of which are resident one or more families, each occupying a separate suite of rooms, entered from a common landing or lobby. The dwelling place of each family is known as a "house." In England the terms are reversed. It is in the former sense that the terms tenement and house are used throughout this investigation. The inquiry was carried out along three main lines.

The Incidence of Measles in Working Class Tenement Buildings.

Life in tenement buildings is, to say the least of it, congested. There is little privacy; the common water closet and the common closes and landings bring together a number of families in the most intimate way. From the epidemiological standpoint each tenement building may almost be regarded as a household in itself. In fact in one example quoted during the course of the report in which all the tenement buildings were situated in a short blind street, the street itself, by virtue of its being the common playground for children, may be considered as an epidemiological field unit. The behaviour of measles, however, in these circumstances is not to be compared with its behaviour in better class tenements where isolation is greater and segregation of families more complete. It was noted during the course of the epidemic that measles was not regarded as a disease worth troubling about. During the period of the invasion children who were not too obviously ill, were not confined to the house. Children with a definite rash were sometimes found sitting on the common stair.

The first example quoted is a tenement building consisting of four storeys, containing in all fifteen houses with one shop on the ground floor. Each house contained one or two apartments. There was a single entry leading through from the street to the back court. On each storey there was one water closet which was used in common. The total population in this building was 68, children under ten years of age numbered 18 and children under ten who had not had measles, numbered ten; they were distributed through seven families. Of the ten children in the building who had never had measles, nine contracted the disease. Two of these were school children and seven were not. The child who did not take measles, was two years of age and lived on the ground floor. There were no other children on the ground floor. The landing between the second and third storey was used as a common playground. The infection was introduced into the building by a school child. This child was removed to hospital on the day following the appearance of the rash and infected seven children under school age and possibly also another school child. In all six families were infected from the one child.

In the second example quoted a double tenement building had a common entry through a pend close. The building was surrounded by business premises and was isolated from other domestic dwellings. It consisted of four storeys with two common stairs, "A" stair being entered from the pend close and "B" stair from the back court. From "A" stair fourteen houses were entered and two were on the ground flat. Facing the street three other houses were entered by front doors, but in none of these did any case of measles occur. The bottom storey of that part of the building served by "B" stair consisted of licensed premises and dairy premises which were entered directly off the street. There were four houses on each of the three upper storeys served by "B" stairs. A single water closet on each storey was used in common by the four families on that storey and the houses consisted of one and two apartments. The total population of the houses approached by "A" stair was 85, children under ten years of age number 24 and children under ten who had not suffered from measles numbered 11. The figures for the houses approached by "B" stair were 63, 19 and 11. The total number of children in the whole tenement building who had not suffered from measles was distributed among six families on each stair. Of the 22 children under ten who had not suffered from measles, 14 contracted the disease. Four of these were school children and ten were not. The first three children affected by the disease lived in the same house. They were all school children and no subsequent infections were referable to them. The infection of one child who did not go to school could not be traced to its source, although twelve days previously two children with which it came into contact, were discharged from hospital. This child infected nine other children who did not go to school and who belonged to five families. Two of these families were on "A" stair living on the same landing and three were on "B" stair, one on the first floor and two on the second. It is pointed out that this tenement building provides an excellent example of how a common entrance to various households by a single close is a considerable factor in facilitating the spread of measles, especially among children under school age.

The third example quoted illustrates the spread of measles through a short street. This street is a *cul-de-sac* bounded by seven tenement buildings. Buildings A, B and C occupied the east side and D, E, F and G the west side. Each tenement building consisted of four storeys and each storey contained four houses of one and two apartments. In tenement B there were only three houses on each storey. The buildings were under observation from October 1, 1925, to May 30, 1926. The total population in these seven buildings was 538. In tenement A there were 58 persons, in B 64, in C 74, in D 82, in E 87, in F 95 and in G 78. There were 173 children under ten years of age. The numbers in each tenement were respectively 16, 20, 26, 33, 33, 34, 11. The children under ten years who had not had measles, numbered 88 and the numbers of these in each tenement were 11, 11, 20, 12, 16, 13, 5. Of the 88 children under ten years of age who had not had measles, 40 contracted the disease. The first three infections in the street were apparently unrelated to one another and the source of infection could not be traced. As far as the street was concerned, they were primary infections. In the course of the inquiry nine such primary infections were discovered. The secondary infections are set out in the report in a table somewhat after the manner of a genealogical tree. The first three primary infections

[1] "An Inquiry into the Relationship between Housing Conditions and the Incidence and Fatality of Measles," by James L. Halliday, M.D., D.P.H.; The Medical Research Council of the Privy Council, 1928, Special Report Series, Number 120.

gave rise to 23 subsequent infections of measles. The time interval between the appearance of the rash varied from ten to fourteen days. The fourth primary infection which occurred in a school child, infected in the class room, was responsible for six subsequent infections in the tenements. One other child infected at school was responsible for only one other infection in the tenements. The main outbreak in the tenements terminated with patient Number 35 who became ill on January 3. Of the last five cases of measles in the tenement (four of the patients became ill in March) four were primary infections.

Special attention is drawn to the fact that although in March the tenements still contained 49 children who had not suffered from measles, only one further case of the disease occurred. It is stated that this may have been related to the chance of exposure. On the other hand, it suggests that the possibility that during the progress of the main outbreak the presumably susceptible children who did not contract measles, were exposed at intervals to small doses of the virus and thus developed a degree of immunity. In the playground of the street and close mouths it is conceivable that infinitestimal doses were received in such a way and at such intervals of time as to build up an immunity after the manner that has been shown by Dudley to occur in diphtheria. In these circumstances, however, such an immunity would be temporary and partial, as the vast majority of individuals is destined sooner or later to undergo an attack of the disease. Dr. Halliday also states that consideration must be given to Brownlee's hypothesis that the cause of a decline of an epidemic is a lessened power of infectivity on the part of the measles virus. Such a view would equally well explain the facts and it is pointed out that the last three patients with primary infections which were introduced into the tenement building of the street, all originated in districts where the epidemic was already on the wane. Dr. Halliday states that the smallness of the samples precludes any definite conclusions being drawn as to the cause of the decline of the measles epidemic, but the findings suggest to him that among the causes of decline is the development of a partial and temporary immunity among the population at risk.

Some interesting observations are made on the order of infection of the tenement buildings. If the main epidemic, that constituted by the first thirty-six cases, is taken into consideration, it is found that the three tenement buildings which were infected at the end of September and early in October provide the largest attack rate, approximately 60% among the children who had not suffered from measles. In the tenement buildings subsequently infected only about 20% of the children who had not had measles, contracted the disease. This fact cannot be related to the season of the year, as during November, December and January measles was at its maximum in other parts of the city. It is tempting to suggest that an explanation may be found in the view that almost as soon as a highly infective case of measles is introduced in one of the buildings in a street, a degree of immunity begins to be set up among all those presumably susceptible persons living in the vicinity. In these circumstances the buildings first affected would have the highest attack rate, while those struck later would be affected to a less degree, as there would be more time available for the development of a process of immunization. On this view it is possible to explain the sudden drop in the attack rate from 56% to 18% between tenement E which was struck on October 4, and tenement A which was struck on October 12, by considering their relative positions in the street. The two tenement buildings which were first affected on September 23, namely B and F, were on opposite sides of the street. Tenement E, infected on October 4, was adjacent to tenement F on the west side and tenement A, infected on October 12, was adjacent to tenement B on the east side. The sudden fall in the attack rate from 56% to 18% occurring between tenement E and tenement A may therefore be attributable to the fact that a longer period of time (nineteen days) elapsed between the infection of A and its neighbour B than the time (eleven days) between the infection of E and its neighbour F. This longer period of time might allow for the development of a greater degree of immunization among the inhabitants of tenement A. At the same time Dr. Halliday states that the samples examined are too small to allow of any definite conclusions being drawn.

The Age Incidence of Measles in Tenement Systems and in Housing Scheme Areas.

A comparison was made between the age incidence of measles in several tenement systems and in housing scheme areas. This comparison was made for control purposes and included thirteen tenement buildings and three housing scheme areas. The time of the inquiry extended from September, 1925, to May, 1926. The tenement building contained 118 houses. The total child population under the age of ten was 284. Children under five years of age numbered 143, the number of cases of measles among these children was 56, a percentage of 39·16. The number of children between the ages of five and ten in the tenement buildings was 141. The number of cases of measles among this number was 18, a percentage rate of 12·76. The housing scheme areas contained 2,067 houses. The total child population under the age of ten was 1,761. The houses were built in blocks of two, four and six. Each house had its own outside door of entry and its own garden. Many of the gardens were separated from one another by railings and hedges. The children under the age of five years numbered 978, the number of cases of measles among them was 76, a percentage of 7·7. The number of children between the ages of five and ten was 783, the number of cases of measles among them was 144, a percentage rate of 18·4. As the official school age is five years, the result may be expressed by the statement that in the tenements examined the incidence rate of measles was 3·7 times greater among children below school age than among school children between the ages of five and ten. In the housing scheme areas, on the other hand, the incidence rates of measles among children below school age was only 0·4 times as great as among school children between the ages of five and ten. This comparison is regarded as providing additional evidence that the tenement system where it involves the grouping of small houses round a common close or stair is responsible for an age shift of measles to infants and young children as opposed to what happens in self-contained houses where the disease tends to be one of school age.

Observations in Schools in a Tenement Quarter and in a Housing Scheme Area.

A comparison is made between children of five and six years of age who have had measles and those who have not had measles in schools situated in a tenement quarter and in a housing scheme area. The inquiry involved four affected schools. Two of them were in working-class tenement districts; in these it was found that the average percentages of children of five and six years of age who had not had measles, were 40 and 38. In the school which supplied a mixed tenement and housing scheme area, the average percentage of children of five and six years of age who had not had measles, was 47. In the school which supplied only a middle-class housing scheme district, the average percentage of those of five and six years of age who had not had measles, was 67·5. The details of this investigation are set out in tables. The relatively small number of those who had not had measles among the infant classes in the schools supplying the tenement districts, is regarded as a further proof of the influence of the tenement building in bringing about an early exposure to the infection of measles. It is seen that not every child who had not had measles, contracted the disease when the schools became affected. Among the infant classes the percentage of presumably susceptible children taking measles showed extraordinary variations, ranging in individual classes from 40 to 95. The average percentage for infant classes over the four schools was, however, 74. In the figures for one complete school it is seen that among the junior school children of seven, eight and nine years of age the attack rate of the presumably susceptible was only 50. In the senior school, composed of children of ten, eleven and twelve years of age, the average percentage of the presumably susceptible who contracted the disease in the classes affected fell to 37. Owing to the wide fluctuations among

the various classes in the percentages of the presumably susceptible who were attacked, it is not easy to give an adequate interpretation of the results, but the average percentages appear to indicate that among the senior classes is to be found a relatively larger proportion of naturally immune individuals. The main feature of the tables presented is that among the infant school classes connected with the housing scheme the largest percentage of children who have not had measles, was found.

The Incidence of Measles as Revealed by Notification.

Dr. Halliday examines two series of notification figures. He again draws attention to the reasons given by him in the introduction why figures obtained by notification should not be taken as representing the true state of affairs. At the same time when the figures are taken as they stand, they lead to conclusions which confirm those arrived at by the field inquiry of which details have just been given.

In the first place, a comparison is made of the age incidence of measles in the poorer and in the residential districts of Glasgow. For this purpose the wards of the city were grouped into four districts, reflecting the social conditions of the inhabitants. There are no wards in Glasgow which do not contain tenements, but among the "residential" wards there are more self-contained houses and relatively fewer tenements than in other districts. At the other extreme the "industrial and poor" districts consist almost entirely of tenements. The table which has been compiled, shows that for each age group up to five years the percentage of individuals infected for each district rises from a minimum in the residential areas to a maximum in the poorer areas. At the age of five a dramatic change takes place and the order is reversed. A possible explanation for these figures is that most of the children in the residential areas escape exposure until they attend school.

The Effect of a Low Age Incidence on Mortality.

Measles is most fatal during the earlier years of life and it kills mainly by means of secondary infection. A study of hospital records in Glasgow during 1925 and 1926 showed that secondary pneumonia and enteritis either singly or in combination were responsible for 90% of the mortality. The comparative case mortality of measles in various age groups cannot be given a definite figure. A table has been compiled in regard to areas where compulsory notification existed. It shows that if the case mortality in the age group five to ten years is considered as unity, the case mortality for the age groups under two years is 21 to 23 times greater. The correspondence between the case mortalities of Aberdeen, Birmingham and Renfrewshire in this respect is remarkable. Reference is made to the question of the correctness and incorrectness of death certificates and several authors are quoted. It is concluded that it is impossible to be definite on the point as to whether the relative case mortality for each age group under five years, compared with that of ages five to ten years, is an over-estimate or an under-estimate.

The Mortality in Tenement and Non-tenement Districts Compared.

It happened that no deaths occurred from measles either in the tenements or in the housing scheme areas which were selected for this special inquiry in Glasgow. The only available data for a comparison of the mortality in tenement and non-tenement districts were the deaths from measles occurring among populations housed generally in tenements, as in Glasgow or in self-contained houses, as in Birmingham. A comparison of the total deaths from measles according to age groups supports the finding that the low age of incidence associated with tenements is of importance. In the tenement cities a greater proportion of deaths from measles occurs under five years of age than is found in non-tenement cities.

The Influence of the Tenement as a Factor in Mortality.

Age is only one of the many variable factors which affect the death rate from measles. Other and probably more important factors include the previous physique of the child, the facilities for adequate ventilation and the all-important personal factor of parental efficiency, which includes the knowledge of the seriousness of the disease, its nursing and its dieting. The part played by these social and environmental factors could not be estimated in this inquiry owing to lack of statistical data. Dr. Halliday thinks, however, that they operate independently of age.

British Medical Association News.

SCIENTIFIC.

THE first quarterly meeting of the Obstetrical Committee of the Obstetrical Section of the Queensland Branch of the British Medical Association was held at the B.M.A. Building, Adelaide Street, Brisbane, on February 24, 1928, DR. D. GIFFORD CROLL, C.B.E., the President in the chair.

Dr. Croll gave a short history of the formation of the Obstetrical Section and said he hoped it would attract those who had not yet joined. He pointed out the value that the use of the form provided by the Section for recording cases, would have in improving obstetrical practice. He said that the form as at present was a beginning and it was hoped that it would be improved as time went on and experience showed what was essential. He was in favour of a comparatively small membership with detailed records of cases rather than a large membership with less detail. He said that members need have no fear of their conduct of cases being criticized, for in the first place the idea of the meetings was discussion, not criticism and in the second place the pratitioner's name was not made known when a case was under discussion and it was obliterated from the record before the same was filed. He extended an invitation to those not members of the Section to send in records of cases of interest which might be read at the meetings. He then said a few words in appreciation of the work of the Statistical Committee and especially of that of Dr. H. W. Tilling.

It may be stated for the information of readers that the Obstetrical Section of the Branch was formed to raise the standard of obstetric treatment in Queensland and to collect accurate statistics. The Section holds one meeting *per annum* at the end of the year. A report of the year's work is presented at this meeting. All members of the Branch are invited to attend the annual meeting. The Obstetrical Committee is composed of those members of the Section who have signed an agreement to record on the forms provided by the Section full particulars of every obstetrical case (by this is meant every viable child) attended in the course of private practice and to submit these forms every month to the Secretary. If no confinements are attended during the month, a return stating this fact must be submitted. The Committee meets every quarter and a report of the records made by members during the previous quarter is presented. A discussion on matters arising out of the reports takes place.

The controlling body of the Section is known as the Executive and consists of the President, the Treasurer, the Secretary and two members of the Committee appointed by election. The Statistical Committee consists of three members of the Executive who are appointed to analyse the cases recorded and to draw up the report for the quarterly meetings of the Obstetrical Committee.

The form supplied by the Section is a comprehensive document. The practitioner's name is placed on top. The patient's initials and her age are given, together with the estimated date of the confinement, the date of the last menstrual periods and the number of the pregnancy. The number of miscarriages is stated, with the date of the first and of the last and the reputed cause. The previous obstetrical history and the general physical condition of the patient are described. The results of antenatal examination are given: the interspinous measurement, the intercristal measurement and the external conjugate; the results of the examination of the urine are stated. As far as the seventh, eighth and ninth month are concerned, the presentation and position of the fœtus must be recorded, the condition of the fœtal heart is noted and the prognosis is stated. Any complications noted during the course of pregnancy are recorded.

The particulars of any treatment before or during labour are enumerated, particularly in regard to such questions as the administration of "Pituitrin," ergot and anæsthetics. In regard to labour itself the date and hour are given of the commencement of labour, of the rupture of the membranes, of the beginning of the second stage, of the birth of the child and of the expulsion of the placenta. The presentation on delivery is stated as well as the sex, age, weight and condition of the child. The delivery, whether natural, by forceps or otherwise, is recorded. The condition of the perineum is described and the numbers of sutures are stated. The expulsion of the placenta is described and condition of the placenta and membranes is recorded. Reasons have to be given for instrumental delivery and abnormalities of the child or of the labour are stated. Full particulars of the puerperium are given—the temperature, the pulse, lochia and fundus and so forth are described.

Space is left for the recording of the results of post-natal examination and the condition of the perineum, cervix and fundus must be stated. Lactation is recorded Finally there is space for the environment to be stated—home, maternity hospital or hospital.

Thymus Glands From Newly-born Children.

DR. F. A. HOPE MICHÔD showed specimens of four thymus glands he had secured from newly-born children. One of these was enlarged, particularly the cervical portion which was hard and surrounded the trachea on each side. It had been removed from a child which had died on the sixth day after birth. At birth the child had been very cyanosed and during the six days of its life there had been no air entry into the upper lobes of the lungs and very little into the lower. The child had had several attacks of dyspnœa and convulsions had supervened just before death.

A discussion then took place as to the treatment of the condition.

DR. C. TUCKER mentioned a thymus gland which he had been able to procure recently. There was practically no cervical portion of this gland, but the lower portion was definite. It was lying on the pericardium and descended about half way down the length of that structure. The posterior surface of the gland was in relation to the superior *vena cava* and aorta. It had not been difficult to dissect away but he doubted the success of attempting surgical removal.

DR. E. S. MEYERS thought that tracheotomy would be useless in the treatment of compression of the trachea in newly-born children from enlargement of the thymus gland. He suggested the use of an intratracheal apparatus and the passing of an intratracheal catheter or tube down the trachea and the leaving of it in position for a few days to tide the child over the critical period.

Analysis of Cases.

The analysis of the ninety-five cases attended by members of the Committee during the first quarter was then presented.

DR. D. GIFFORD CROLL congratulated the Statistical Committee on their efforts and a discussion of the analyses then followed.

DR. A. C. F. HALFORD made some remarks on the importance to be attached to the rate of the fœtal heart during labour. He thought that it could be accelerated to 160 beats per minute without concern, but that if the increase in rate continued, he noticed that a drop in the rate followed and when that occurred he expected that the child would be difficult to resuscitate after birth. He thought that the fœtal heart was difficult to stop and that stoppage occurred only in conditions presenting extreme obstruction to the fœtal circulation. He thought the tenacity of life of the fœtus was extraordinary.

DR. M. ELLIOTT SMITH asked why uterine inertia was given as a reason for the use of instruments. He would like to know if the inertia were primary or secondary and if secondary, he thought that it was a contraindication to the use of forceps.

DR. M. GRAHAM SUTTON remarked on a case he had recently attended in which the child was born to the umbilicus in a breech presentation by the time he arrived on the scene. Both the arms had been extended. They had eventually been brought down and the child had been born. The fœtal heart had been beating rapidly at birth, but the child had made no attempt to breathe. At first the heart rate had been rapid and the child cyanosed. It had then become pallid, the fœtal heart had slowed and become irregular and the child had died in a convulsive spasm.

Dr. Meyers remarked on the forceps rate of 17%. In the records of his own two hundred cases the forceps rate was 12%. The forceps rate at the Lady Bowen Midwifery Hospital at the same time was 5%. He thought that with patience he might have reduced his percentage. In regard to still-birth, he thought that injury to the child frequently caused death.

DR. H. STANLEY WATERS quoted the opinion of some authorities on still-births. After breech deliveries they thought that the death was due to tentorial tears and that breech deliveries should not be hurried. He thought that cerebral hæmorrhage and cerebral compression might have accounted for the still-birth in Dr. Sutton's case.

Dr. Croll thought that the high percentage of still-births in the series was due to the small number of cases. In regard to the morbidity rate of the general hospitals which showed at 12%, he thought this was high, because patients with abnormal conditions were sent into those hospitals for treatment.

DR. K. WILSON commented on the number of occipito-posterior presentations that had been rotated with instruments before delivery. He thought that manual rotation of the head accompanied by rotation of the shoulder with the hand inside the uterus and then delivery by the application of axis-traction forceps with the occiput anterior, was the easiest method. He had tried rotating the head with Kielland forceps and then extracting, but preferred the former procedure.

DR. G. A. MCLEAN was in favour of rotation of the head by means of Kielland forceps and then extracting. He had found it easy of accomplishment, but did not favour turning the head with axis-traction forceps.

DR. A. C. F. HALFORD was not in favour of instrumental rotation of the head. He preferred manual rotation and then the application of forceps.

DR. C. E. TUCKER said he had had very many occipito-posterior positions in his practice. He laid much stress on the importance of turning the shoulders as well as the head. He did not like the method of rotating the head with instruments. He thought that a lot of occipito-posterior fœtuses, if left to Nature, would rotate in an anterior direction.

DR. HEDLEY BROWN asked at what stage of dilatation of the *os* should rotation be attempted.

Dr. Michôd asked as to whether the fœtal mortality was higher with rotation of the head by instruments. He said that he got good results by manual rotation of the head, application of a binder, narcotic drugs and patience.

Dr. Wilson said that he had had large success with Buist's method of pad and binder in the ante-natal correction of occipito-posterior presentations, but that he was not always successful in recognizing them as such on examination.

Dr. Croll said that in occipito-posterior presentations the cervix usually dilated slowly and the question of the time to interfere depended on the condition of the mother and the child. He had found the pad and binder method useful, but occasionally it failed. He laid stress on the fact that the pad should be hard and small.

DR. L. W. GALL did not agree that it was possible to diagnose persistent occipito-posterior positions before the cervix was fully dilated. To him a persistent occipito-posterior position meant face to pubes and this could not occur until the cervix was fully dilated. He said that in the treatment of persistent occipito-posterior positions he worked with the left hand inside the uterus turning the head and the right hand on the abdomen turning the shoulder. He then applied forceps without withdrawing his left hand.

Several selected case histories from the series were then read to the meeting in detail.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Sangster, John Clive, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.
Hackett, Cecil John, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.
Peters, Geoffrey Ernest, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.
Harbison, Alan Thomas, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.
Dunn, Talbot Lewis, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Baur, Georg, M.D. (Univ. Munich) 1894; State Exam., Certif., (Munich), 1894; 193, Macquarie Street, Sydney.
Byrne, James Michael, M.B., Ch.M., 1926 (Univ. Sydney), Sierra, Holden Street, Ashfield.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Clifton, Vivian Roy, M.B., Ch.M., 1925 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Dick, Junior, Robert, M.B., Ch.B., 1923 (Univ. Edinburgh), F.R.C.S. (Edinburgh), 1926; 7, Benelong Crescent, Bellevue Hill.
Galbraith, Thomas, M.B., 1924 (Univ. Sydney), Royal Hospital for Women, Paddington.
Lynch, Richard Bentley, M.B., Ch.M., 1920 (Univ. Sydney), Emmaville.

Public Health.

SCHOOL INSPECTION IN SOUTH AUSTRALIA.

THE MINISTER OF EDUCATION OF SOUTH AUSTRALIA has issued his report for the year 1927. The report covers every activity of the Department. Among the subjects discussed are the number of schools in operation, school attendance, primary education, secondary education, technical education, the teaching staff and finance. Under the heading of primary education medical and dental inspection receives attention. In an appendix Dr. Gertrude Halley, the Principal Medical Officer, gives many interesting details of the activities of her branch of the Department.

Medical Examination.

All primary and secondary public schools in the State have been visited once and all children attending at the time that the visits were made, have been medically examined. This is in accordance with the scheme of inspection arranged at the inception of the Branch which deals with medical inspection. All children entering high schools and infant schools have been examined, in addition to the children attending from the fourth to the seventh grades in the metropolitan area and the more important country centres. Further, all those in the fourth, seventh and tenth grades in the central schools have been examined. The desire of the Department has thus been fulfilled for a medical examination of children at an interval of every three years during their school life. Some of the small country schools have been visited for the second time and the routine medical examination has been carried out.

The total number of children examined was 24,104. This number is somewhat smaller than in previous years owing to resignations from the medical staff. Defective vision was found in 620 children and defective hearing in 116. Tonsils and adenoids requiring attention were found in 1,138 children. Abnormal conditions of the heart and chest numbered 174 and those of the skin 143. The parents of the children were notified by written notices of these serious defects. Dr. Halley expresses gratification at the fact that the number of notifications to parents is steadily decreasing. She does not give any figures for the previous years by which comparisons could be made. The most suitable way of depicting these results would be to estimate the percentage of defects discovered each year and to plot them in the form of a graph covering the activities of this Branch of the Department since its inception. Many of the children examined during 1927 have been examined for the second time. Most of these children, especially in the metropolitan area, have received attention. Unfortunately when no attention has been given to these physical imperfections, they have become worse. This is especially true in regard to the visual defects. Of the children examined in the schools for infants many have had attention, particularly those suffering from adenoids and enlarged tonsils, before they became enrolled at school. It is claimed that the parents have realized the benefits resulting from treatment given to their elder children and that the talks to mothers have taken effect. In one school 6% of the children had received attention before coming to school.

A special inquiry has been made into the incidence of heart trouble following acute rheumatism; thirty children were discovered suffering from heart disease in these circumstances. South Australia is claimed to compare very favourably with other places in this matter and the suggestion is made that the warm, dry climate is responsible. No tuberculous infections were discovered. The response by parents to the notices which have been sent has on the whole been very satisfactory; 712 children were treated after their parents had received the first notice and 548 received attention after the second "urgent" notice had been sent. The school nurses visited 489 homes in the metropolitan area and 86 in the country and explained to the parents the need for attention. The nurses were well received and in several instances they took children to hospital because the mothers were unable to do so.

Colour-Blindness.

Dr. Christie had continued his research into the incidence of colour-blindness in South Australia. More scientific tests were procured and some original work was done. The standardized tests used were those with Ishihara's plates and Holmgren's wools. In all 1,078 girls and 4,716 boys were tested, a total of 5,794. It was found that eight girls or 0·74% were colour-blind to a limited degree and 308 boys or 6·55% were affected. The examination of the girls revealed the fact that while their powers of discriminating colour-contrast are defective, they do not suffer from the typical colour-blindness exhibited by some males. The latter defect is rarely found in women. All the cases of blindness were of the red-green type, no blue-yellow blindness was detected. In pictures drawn by these children the plants had red leaves and green flowers. Experiments were conducted in an effort to determine what colours colour-blind people see when they look at a coloured object and to find a means of remedying the defect. Various coloured glasses were used. So far no definite results have been achieved, although interesting data have been obtained. The incidence of colour-blindness varies at different ages. It was found that at eight years of age the percentage of those who were colour-blind was 5, at nine years of age it was 6, at ten it was 7, at eleven 10·6, at twelve 6·1. at thirteen 7·2, at fourteen 6·4 and at fifteen 6·7. It is thought that if upon further experiment during the next year the higher percentage remains in the ten to twelve group, the condition may be regarded as having a developmental origin. Further research may then indicate the cause and if so, the defect may be arrested. No colour-blindness was found among the children at the Point McLeay Aboriginal Station.

The incidence of colour-blindness will have an important bearing on the question of vocational guidance. Circulars dealing with colour discrimination have been issued to the children advising them of the various occupations in which they would be seriously handicapped.

Infectious Diseases.

During the year 128 infections of diphtheria have been notified. This number is less than that of 1926. An outbreak of diphtheria occurred at the Richmond School and most of the patients had been taught in the same class room. Swabbings were taken from these children, carriers were detected and after isolation and disinfection the outbreak was stamped out. So far no scheme of immunization against diphtheria has been undertaken in the State.

An epidemic of pertussis occurred during the year throughout South Australia, 1,435 cases being reported. It is pointed out that during the epidemic there were many mild infections which were not reported owing to the fact that the characteristic "whoop" was not pronounced.

Measles took on an epidemic form, 1,678 infections were reported. This outbreak was not nearly so extensive as in 1925, when the notifications numbered 9,447. In 1926 the number was only 74. It is pointed out that pertussis and measles appear to run in cycles and that the gradual spread of both diseases along the various routes of communication can be traced.

Among the other notifications there were 875 cases of mumps, 477 of varicella and 13 of enteric fever.

Dental Treatment.

In the city children under ten years of age only receive treatment at the Dental Clinic; in the country, however, treatment is given to all children. The total number of fillings in permanent teeth during the year was 4,107 and the number in deciduous teeth was 3,802. The number of permanent teeth extracted was 756 and the number of deciduous teeth extracted was 5,347. It is to be hoped that in future years the number of permanent teeth extracted will become less. This question is not discussed, but the explanation most probably lies in the fact that apathy has been shown in this matter not only by the parents of the children, but by the teachers. At the present time it is likely that in many instances teeth are neglected for so long that extraction is the only satisfactory means of treatment.

The Work of the Psychologist.

A lay psychologist is attached to this Branch of the Department and her report is included in that of Dr. Halley. During the year psychological examinations were made of 576 children. It is interesting to note that 33 of these were at the request of the parent or guardian. Four children were examined at the Children's Court. In connexion with this work what are known as opportunity classes, have been formed. At the present time there are twelve such classes in the metropolitan area. Of the 230 children attending 146 are dull and backward, 66 are educable feeble-minded children who require a special school, and 18 are uneducable and will need institutional care and supervision.

During the year the psychologist examined individually all the children attending the school at Minda and saw altogether 209 of the inmates. Of these only 82 are under the age of sixteen years. Of these fifteen are educable, feeble-minded children, 13 are high grade imbeciles and eight low grade imbeciles were among the number. The opinion is expressed that as the school is now constituted it would be impossible to carry out any effective training. There is no proper grading of children, no attempt is made to use any modern methods of dealing wtih such children and there exists no systematic cooperation between home and school.

At the beginning of the year all children entering high schools were subjected to a group test of intelligence. This was done in order to ascertain the relative value of such tests and of the qualifying examination as qualifications for children entering the school. This experiment is to be continued in 1928, when the results will be correlated with the results of the intermediate examination.

A course of ten lectures on the subnormal child has been given to students at the Teachers' College. In this connexion the psychologist remarks that the difficult, nervous, unstable child is often being taught unsuccessfully. Too often instead of being studied and treated according to their needs these children are made more fearful and more emotional by being given corporal punishment. The most pressing need for the subnormal child is necessarily the establishment of a special school or special classes for subnormal children over twelve years of age.

Correspondence.

AN UNUSUAL EPIDEMIC.

SIR: Dr. A. M. Edwards (THE MEDICAL JOURNAL OF AUSTRALIA, May 26, 1928), referring to this epidemic, states: "I have no doubt that the epidemic was dengue." Bearing in mind the severity and uniformity of the symptoms in an epidemic of dengue, it is highly improbable that this disease could occur in an aberrant form; yet Dr. Edwards admits that five out of six of his cases were atypical.

In this district the mildness of the epidemic was one of its salient features.

Undoubtedly there has been a plague of mosquitoes locally, as well as one of "stinging flies." Three years ago the late Dr. E. W. Ferguson carried out an extensive mosquito survey at Narrandera, and in his report he states: "The chief mosquito present is *Culex fatigans*." He found a few specimens of *Culex annulirostris*, and at one point anopheline larvæ. No *Stegomyia fasciata* were found.

Dr. Ferguson's report also states: "*S. fasciata* have not yet been found so far south." In Volume II, Osler's "System of Medicine" (page 138) the approximate southern limit of dengue is given as 23½° south latitude (although the 1926 to 1927 epidemic extended to the vicinity of 32° south).

Both Hay and Narrandera are situated 34° 40″ south latitude.

The same authority quotes from a report by Cleland and Bradley: "Epidemic dengue in Australia is approximately coextensive with the known distribution of the *S. fasciata*. It does not extend beyond the area in which this mosquito is prevalent."

The unusual epidemic (THE MEDICAL JOURNAL OF AUSTRALIA, May 5, 1928) has entirely disappeared with the advent of winter. That either stinging flies or mosquitoes are causative agents may be a moot point, but these insects likewise have gone into winter quarters.

Yours, etc.,

J. R. NIMMO.

Narrandera, New South Wales.
May 29, 1928.

Post-Graduate Work.

LECTURES IN MELBOURNE.

THE MELBOURNE PERMANENT COMMITTEE FOR POST-GRADUATE WORK has decided that the fee for attending the course of lectures by Professor F. R. Fraser from August 7 to August 17, 1928, will be three guineas. Special arrangements will be made for superintendents, registrars and resident medical officers of hospitals.

BIRTHDAY HONOURS.

THE announcement has been made that His Majesty the King has been pleased to confer the honour of knighthood

on Dr. H. Simpson Newland, C.B.E., D.S.O.. Sir Henry's work during the war and his achievements in civil surgery are well known to his colleagues. We offer him the hearty congratulations of the medical profession in Australia.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned has been registered under the provisions of Part I of the *Medical Act*, 1915, of Victoria, as a duly qualified medical practitioner.

Walker, Ellen Sarah, M.B., B.S., 1926 (Univ. Melbourne), 517, Sydney Road, Parkville.

Books Received.

TUBERCULOUS INTOXICATIONS: CONCEALED AND MASKED TUBERCULOSIS: A CLINICAL STUDY, by Joseph Hollós, M.D.; 1928. Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 140. Price: 10s. 6d. net.

OUTLINES OF DENTAL SCIENCE, Volume XI; Dental Metallurgy, by A. Johnstone Brown, L.R.C.P. & S., L.D.S. (Edinburgh); 1928. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 190, with illustrations. Price: 7s. 6d. net.

SOME MORE MEDICAL VIEWS ON BIRTH CONTROL, Edited by Norman Haire, Ch.M., M.B.; 1928. London: Cecil Palmer. Post 8vo., pp. 239. Price: 7s. 6d. net.

Diary for the Month.

JUNE 12.—Tasmanian Branch, B.M.A.: Branch.
JUNE 12.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 14.—Victorian Branch, B.M.A.: Council.
JUNE 14.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 18.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 19.—Tasmanian Branch, B.M.A.: Council.
JUNE 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 20.—Western Australian Branch, B.M.A.: Branch.
JUNE 20.—Central Northern Medical Association, New South Wales.
JUNE 20.—Western Medical Association (Dubbo), New South Wales.
JUNE 20.—Section of Obstetrics and Gynæcology, New South Wales Branch, B.M.A.
JUNE 22.—Queensland Branch, B.M.A.: Council.
JUNE 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. Charles Sutherland (B.M.A.) has been appointed Certifying Medical Practitioner at Melbourne.

* * * *

Dr. Oswald Joynt (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for Insane and Receiving House, Royal Park, Victoria.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Radiologist, Pathologist and Registrar.
STATE PUBLIC SERVICE, QUEENSLAND: Second Assistant Medical Superintendent.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY: Honorary Assistant Anæsthetist, Relieving Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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